Astrovirus infection in hatchling turkeys

The economic impact isn't known, but losses do stem from high morbidity. Growth is decreased and flock unevenness follows.

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Astrovirus is just one of several different enteric viruses identified in association with turkey viral enteritis, which has been a primary concern in the area of enteric diseases of turkeys, although the economic impact on the turkey industry is not fully known.[1] Losses stem from the high morbidity of the disease resulting in decreased growth and flock unevenness. Additionally, this disease appears to be highly prevalent and geographically widespread. Astrovirus causes problems even for producers who employ good management practices in modern facilities.

Studies in the past ten years have identified several enteric viruses in association with this disease. These viruses are rotaviruses, groups A and D;[9,10,11,13,14] astrovirus,[6,7,10,11] enterovirus,[7,8,9] parvovirus,[12] and reovirus[2,4,7,10]. Their individual roles in turkey viral enteritis have not been fully delineated.

Previous surveys found that astrovirus and group D rotavirus occur more frequently in turkey poults with clinical signs of turkey viral enteritis.[8,10] Natural infections with astrovirus, in avian species, appear to occur only in turkeys.[3] However, only rarely is astrovirus found as a single-virus infection.[7,8,10] Astrovirus infections occur in poults from hatch to four weeks of age. The incubation period is short, 48 to 72 hours, and duration of the disease is 10 to 14 days.

High morbidity

Clinical signs are variable in expression and severity but, typically include diarrhea and listlessness. The disease is characterized by high morbidity and low mortality. Characteristic post mortem lesions consist of large, gaseous dilatated ceca filled with yellow-brown frothy material, generalized loss of intestinal tone, and intestinal walls are flaccid and translucent.[6,11] Currently, diagnosis is based on the presence of typical clinical signs, necropsy lesions and identification of astrovirus by negative stain immune electron microscopy (IEM).[3,5,6,10]

Astrovirus is typically described as a small round virus 28-30 nm (ave 29.6 nm) but, only a small percentage of virions exhibit the star-like surface morphology from which the virus derives its name.[3,6] The primary criteria for identification is based on ultrastructural characteristics noted on direct electron microscopy (negative staining EM). However, without characteristic star-virions identification is difficult.[5] Consequently, other methods are commonly used to verify the presence of astrovirus including Cesium chloride (CsCl) isopyknic centrifugation, immunofluorescence and IEM (negative stain IEM).

Limited study reports

There are limited reports of pathogenicity studies of these two viruses. These studies used specific pathogen free poults inoculated with either group D rotavirus,[9,11] astrovirus,[6] or the two viruses combined[9,11]. Both, the individual viruses and the combination of viruses produced typical signs and gross lesions. Additionally, statistically significant differences in weight and malabsorption were documented via d- xylose absorption in the astrovirus inoculated poults.[6]

Our research addresses several unanswered questions pertaining to the role of astrovirus as an enteropathogen. (1) Can astrovirus infection be induced in commercial turkeys under experimental conditions? (2) Does experimental astrovirus infection produce histologic, morphometric or ultrastructural changes in the small intestine? (3) Does astrovirus exhibit specific regional or intestinal cell tropism? (4) Does astrovirus infection cause maldigestion?

The specific methods used in these studies were light microscopy to document histologic lesions; computerized image analysis, to identify morphometric changes; electron microscopy, to document lesions at ultrastructural level; and intestinal mucosal disaccharidase analysis, to evaluate carbohydrate digestive function in the small intestine.

In four separate experiments, two and five day old commercial poults were inoculated orally with astrovirus. Poults were examined on various days

post inoculation (PI) ranging from 0.5 to 14 days. Astrovirus was identified by IEM, in pooled intestinal contents from inoculated poults, but not from controls. All poults were negative for salmonellae and Arizona species. No other viruses or recognized enteroprathogens were detected in either inoculated or control birds.

Clinical signs noted

Clinical signs and gross lesions were similar to those reported by Reynolds et al.[6] Poults on days two through 10 post-inoculation had diarrhea, generalized loss of intestinal tone and dilatated ceca containing light yellow to tan fluid to semi-solid feces and gas. Mild crypt hyperplasia was noted one day post-inoculation in the proximal jejunum and by day three the distal jejunum and ileum were documented by image analysis on day three post- inoculation. The entire small intestine is affected seven days after inoculation. Significant morphometric changes noted at day seven post-inoculation involve the distal small intestine but are variable.

Virus particles were found in a few villous enterocytes in the ileum and distal jejunum, on days two and three and day two PI, respectively. The scattered infected cells were located on the sides or near the base of villi. Electron dense viral aggregates found two days after inoculation were located in and around dilated cytocavitary spaces. Virus particles were arranged in quasi-crystalline or crystalline arrays and also as ovoid viral aggregates. Release of viral aggregates into the intestinal lumen was seen on rare occasion in the distal jejunum three days PI.

Intestinal disaccharidase activity, specifically maltase, was decreased ($P<0.5$) throughout the entire small intestine from three to seven days PI. Ten days after inoculation no significant difference was detected in specific maltase activity in inoculated versus control poults. At 14 days post- inoculation specific maltase activity was increased ($P<0.5$) in all sections of the small intestine of inoculated poults except the proximal jejunum.

Conclusions reported

In conclusion, astrovirus alone is an effective enteropathogen in hatchling turkey poults. Astrovirus produces enteric disease with typical clinical signs, gross and subtle microscopic lesions and replicates in villous enterocytes of the distal small intestine.

Furthermore, astrovirus causes a transient period of maldigestion by decreasing intestinal disaccharidase activity throughout the majority of the small intestine. Dysfunction of the intestinal mucosal disaccharidases would augment concurrent malabsorption, if this occurs in commercial poults, as previously documented in SPF poults[6].

Maldigestion and malabsorption together could produce the diarrheic condition and resultant "stunting" seen in astrovirus infected poults. The primary pathogenic effect of experimental astrovirus infection in hatching turkey poults appears to be at the biochemical level, primarily affecting disaccharidase activity, rather than producing histologic lesions. —*Proc. Iowa Poultry Symposium, April 9, 1992, Iowa State University.* ❐

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CURRENT EGG PACKAGING being offered is not conducive to reducing school labor costs. The egg industry needs to take the initiative and find better methods of packaging or to coordinate manufacturing firms into developing egg products that the schools can readily use—*News from the California Egg Commission.*

Correction

The January issue of Poultry Digest featured a story titled "Ascites syndrome: overview and update," by Ted W. Odom, Texas A&M University. The author's name was spelled incorrectly, an error which must be blamed on the editor, who was obviously out to lunch the day the copy was handled. We do apologize.