

ESCHERICHIA COLI SEPTICEMIA IN PHEASANTS

Authors: CREITZ, J. R., and SMALL, NOLA N.

Source: Bulletin of the Wildlife Disease Association, 3(2) : 68-69

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-3.2.68>

The BioOne Digital Library (<https://bioone.org/>) provides worldwide distribution for more than 580 journals and eBooks from BioOne's community of over 150 nonprofit societies, research institutions, and university presses in the biological, ecological, and environmental sciences. The BioOne Digital Library encompasses the flagship aggregation BioOne Complete (<https://bioone.org/subscribe>), the BioOne Complete Archive (<https://bioone.org/archive>), and the BioOne eBooks program offerings ESA eBook Collection (<https://bioone.org/esa-ebooks>) and CSIRO Publishing BioSelect Collection (<https://bioone.org/csiro-ebooks>).

Your use of this PDF, the BioOne Digital Library, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Digital Library content is strictly limited to personal, educational, and non-commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne is an innovative nonprofit that sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

BRIEF NOTES, SURVEYS, AND COMMENTS

ESCHERICHIA COLI SEPTICEMIA IN PHEASANTS

Escherichia coli is often isolated from materials associated with disease. However, this ever-present organism is usually considered to be a contaminant or a secondary invader (Biester and Schwarte, 1965). The present findings demonstrate its role as a primary invader. It is believed that this is the first report of *Escherichia coli* septicemia in pheasants.

Since pheasants had been incriminated as a virus reservoir in an epidemic of Eastern Equine Encephalitis in 1959, it was theorized that pheasants might be a good indicator species reflecting the presence of EEE Virus in local fauna (Scatterday and Hart, *Morbidity and Mortality Weekly Report*, USPHS, Vol. 10, No. 45, 1961). A flock of pheasants was maintained at a bird farm operated by the Rod and Gun Club. Husbandryman at the farm had been instructed to contact the Post Veterinarian immediately if disease occurred in the pheasants. On July 31, 1961, the veterinarian was notified that nine of 300 four-month-old pheasants had died during the night. The sick birds showed no signs or symptoms before death except weakness. The pheasants were being raised under good conditions with modern poultry equipment. Only commercial poultry feed for the appropriate age birds was used. Minor parasite problems had been encountered in previous years, but no difficulties had been experienced with this group of birds.

Two of the nine dead birds were autopsied at the pen. Congestion was noted in the lungs of one, the other bird showed no unusual postmortum findings. A moribund bird was brought to the laboratory where it was autopsied under aseptic conditions. The only ab-

normal gross finding was congested lungs. Portions of the brain were macerated in buffered saline and 0.03 ml portions were injected intracerebrally into $\frac{1}{2}$ day old and adult mice in an attempt to isolate and help differentiate a possible virus infection. Thioglycollate broth was also inoculated with aliquots of the brain suspension to detect possible bacterial contamination. Examination of the cecal and intestinal contents for parasites revealed only occasional coccidia.

Microscopic examination of heart blood showed bacterial rods outnumbering the blood cells 6:1. Further examination indicated that the bacteria were Gram negative and non-motile. On the basis of this finding, oxytetracycline was immediately added to the feed of the live birds and another sick bird was requested to confirm the septicemia. The fourth bird autopsied showed no unusual gross findings. Aseptically drawn heart blood was inoculated onto various bacteriological media, including media for enteric organisms. Microscopic examination of the blood revealed similar organisms in about the same numbers as were found in the previously examined blood. All 16 bacterial cultures of heart blood and brain tissue suspensions produced pure, heavy growths of motile, biochemically typical *Escherichia coli*. The organism was not typable with locally available antisera, but was later typed as *E. coli* O1:ab:K1:H7.

Initial sensitivity studies indicated the organism was sensitive to oxytetracycline, tetracycline, chloramphenicol, streptomycin, and neomycin. Quantitative tube sensitivity studies showed the organism to be sensitive to 0.78 mcg/ml of oxytetracycline and to 25 mcg/ml of dihydrostreptomycin.

Seven of the suckling mice inoculated with brain suspension died on the second

day after inoculation. The eighth suckling mouse died on the fourth day. Of the six adult mice, one died on the first, two died on the second, and one each on the third and fourth day. *Escherichia coli* was recovered from the brains and blood of all the dead mice. The remaining adult mouse was sacrificed in good health after surviving the inoculation 28 days.

The early observation that the organism was non-motile in the birds and mice and motile in culture was reexamined. When *E. coli* suspensions from culture were inoculated intraperitoneally into mice, the organism became non-motile, both before and after death of the mice. Two cycles of alternate passage between mice and culture always resulted in the organism being non-motile in mice and motile in culture. The high degree of pathogenicity for mice initially demonstrated by the organism in the brain suspension was also substantiated. The mice, which were intraperitoneally inoculated with culture suspension, all died or were moribund and were sacrificed in two days. Overwhelming numbers of *Escherichia coli* were demonstrated microscopically and culturally in the heart blood of these intraperitoneally inoculated mice.

After oxytetracycline was added to the feed of the pheasants, no additional deaths occurred and a marked improvement was noted in the flock. No other disease problems were observed in this group of birds. The source of the infection was not determined.

Summary

Signs of colibacillosis were observed in a flock of captive pheasants. A diagnosis of *Escherichia coli* septicemia was made on two moribund pheasants after large numbers of this bacterium were observed microscopically in their blood and the organisms were recovered on culture of the blood. The serotype of the organism was O1:ab:K1:H7. Marked improvement of the sick birds was

noted after they were given oxytetracycline in the feed.

Acknowledgement

The authors wish to thank Dr. William H. Ewing of the Communicable Disease Center, Atlanta, Georgia, for sero-typing the organism. The cooperation and effective control measures instituted by Captain Sam E. Levington, VC, Acting Post Veterinarian, were appreciated by all concerned.

J. R. CREITZ
and NOLA N. SMALL

Microbiology Dept.
1st U. S. Army Medical Lab No. 1
Ft. Meade, Md. 20755
27 October, 1966

THE OCCURRENCE OF HEMATOZOA IN ROBINS OF CENTRAL WASHINGTON

Despite the wide distribution and abundance of robins, their parasitology has received very little study (Manwell, 1955, J. Protozool., 2: 85-88). Work on avian blood parasites in Washington is limited apparently to a study in magpies and english sparrows of eastern Washington (Wagner, 1946, Birdbanding, 17(2): 53-55). Blood and tissue smears were taken from nestlings, juveniles and adult robins (*Turdus migratorius*) collected in central Washington from fall 1965 to fall 1966. The term "juvenile", in this study, was applied to those robins that were capable of flying and still retained spots on their breasts. Nestlings were those birds still remaining in the nest proper. Juveniles and adult birds were shot and as soon as possible after death, thin blood films were made from large thoracic vessels or directly from the heart. Tissue smears were made from small sections of lung and kidney. Prior to making the smear, each piece of tissue was wiped clean of blood with cheese cloth, macerated on the slide and then all but a thin smear was removed. Portions of spleen, heart, small intestine, brain, kidney, lungs and liver were