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Authors: SCHNEIDER, WARREN J., and HASLETT, THERESA M.

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ATTEMPTED EXPERIMENTAL TRANSMISSION OF PSEUDORABIES VIRUS TO EUROPEAN STARLINGS *(Sturnus vulgaris)*

WARREN J. SCHNEIDER and THERESA M. HASLETT, School of Pharmacy, University of the Pacific, Stockton, California 95211, USA.

Abstract: Forty European starlings *(Sturnus vulgaris)* were inoculated orally with pseudorabies virus. Subsequent attempts to isolate the virus from feces, liver, lung and kidney of the birds were negative; antibody against the virus could not be detected in their serums.

INTRODUCTION

The European starling *(Sturnus vulgaris)* was introduced into the United States in 1890, when 60 imported birds were released in Central Park, New York City.[4] Forty more birds were released there in 1891. The vast hordes of European starlings now occupying the United States descended from these 100 birds. According to a recent 5-year nation-wide roost survey, an estimated half-billion starlings and blackbirds are now in the continental United States.[14]

Starlings cause huge economic losses when they descend upon orchards, vineyards and feedlots to satisfy their appetites.[2,7,16] The enormous populations of free-ranging starlings and their tendency to overlap with the environment of man also leads to speculation regarding their potential role as carriers of disease. Several investigators have reported finding starlings infected with tuberculosis.[3,11,12,18] Wild birds, particularly starlings, have been suspected of carrying foot-and-mouth disease virus from continental Europe to the British Isles.[13] Starlings have been associated with epidemics of histoplasmosis in Iowa,[5] Michigan[6] and at Ft. Campbell, Kentucky.[17] In an experiment conducted in Indiana, suspensions of intestines from hogs infected with transmissable gastroenteritis were fed to starlings.[15] Feces voided by these starlings, 1, 2, 4, 8, 16 and 32 h. after ingestion of the infected intestines were fed to specific pathogen free pigs, and induced the disease in these animals.

Large numbers of starlings were observed on a California ranch where more than 1,600 garbage-fed pigs died of pseudorabies (PR) during a 3-year period.[10] It was postulated that the birds may ingest PR virus-contaminated nasal secretions of pigs, then fly to other livestock premises and shed the virus in their feces. Alternatively, the birds may actually become infected with the PR virus and then shed it. The experiment reported here was designed to test these hypotheses.

MATERIALS AND METHODS

Fifty adult native starlings[1] were alloted to 2 groups — 40 principals (group 1) and 10 controls (group 2). Each of the 40 birds in group 1 was inoculated orally with 0.3 ml of PR virus suspension containing $10^{7.24}$ $TC1D_{50}$ per 0.2 ml.

Pooled feces from each group of birds were collected at 0, 1, 3, 8, 16, 24, 32, 40 and 96 h. after inoculation of virus into

[1] Starlings were supplied by Dr. R. G. Schwab, Assistant Wildlife Biologist, Department of Animal Physiology, University of California, Davis.

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the principals. Each fecal sample was thoroughly mixed, using the collection swab, placed in a tube with nutrient media and antibiotics, and inoculated into cell cultures. The cultures were examined daily up to seven days for cytopathic change.

Before the starlings were allotted to the two groups, blood samples from 5 birds were collected, pooled and determined to be free of antibodies against PR virus. Blood samples were obtained from 5 birds in group 1 14 days after inoculation and tested for antibodies against PR virus. Antibody determinations were conducted using a serum neutralization test described elsewhere.[9]

Groups of 5 birds from group 1 were killed at 8, 24, 48 and 96 h. after inoculation. Specimens of liver, lung, and kidney were obtained from each bird and pooled by organ for each time interval. Each pooled sample of necropsy material was inoculated into cell cultures and observed for cytopathic change daily for 7 days.

Upon completion of the PR virus transmission experiment, the authors wished to determine the average pH of starling gizzard contents because of possible adverse effects on viral infectivity. The gizzards of 10 starlings, collected as described elsewhere,[8] were removed and the pH of the contents was determined using general range (1.0 - 14.0) pH paper.

RESULTS

Attempts to isolate PR virus from the feces and tissues of adult starlings that had been inoculated with the virus were all negative. Antibodies against PR virus were not detected in the serum of the experimental birds after inoculation of the virus.

The average pH of gizzard contents taken from 10 fledgling starlings was 3.6.

DISCUSSION

The negative results indicate that concentrated PR virus introduced directly into the oral cavity of starlings does not survive long enough to be passed with the feces. In addition, the virus apparently does not replicate within cells of the various starling tissues from which isolations were attempted. The virus infectivity was probably destroyed by the low gizzard pH, since a pH<5 adversely affects PR virus.[1]

The negative results do not discount the possibility that starlings might carry PR virus from infected premises externally on their feathers, feet or other body parts, thus acting as passive carriers. The ability of this virus to exist outside of a host was demonstrated by its survival on hay for 30 days in summer and 46 days in winter.[1]

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LITERATURE CITED

1. ANDREWS, C. 1964. *Viruses of Vertebrates.* The Williams and Wilkins Co. 220.
2. At last you can control starlings. 1968: Farm Jour. 92: 64J.
3. BICKFORD, A.A., G.H. ELLIS and H.E. MOSES. 1966. Epizootiology of tuberculosis in starlings. J. Am. vet. med. Ass. 312-318.
4. CHAPMAN, F.M. 1939, republished 1966. *Handbook of Birds of Eastern North America.* 2nd ed. Dover Publications, Inc., New York City.

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5. DIESCH, S.K. and K.R. LONG. 1967. Starlings and health. Iowa Bird Life, 37: 45-48.
6. DODGE, H.J. 1965. The association of a bird-roosting site with infection of school children by *Histoplasma capsulatum*. Am. J. Pub. Hlth. 55: 1203-1211.
7. FOWLER, R.G. 1967. New weapon for starling sufferers. Farm Jour. 91: 45H.
8. HASLETT, T.M. and W.J. SCHNEIDER. 1977. Occurrence and transmission of *Toxoplasma gondii* in European starlings *(Sturnus vulgaris)*. J. Wildl. Dis. 14: 173-175.
9. HOWARTH, J.A. 1968. A serological study of pseudorabies in swine in California. J. Am. vet. med. Ass., 152: 1114-1118.
10. HOWARTH, J.A. and A. DePAOLI. 1968. An enzootic of pseudorabies in swine in California. J. Am. vet. med. Ass., 152: 1113-1118.
11. KEYMER, I.F. 1958. A survey and review of the causes of mortality in British birds and the significance of wild birds as disseminators of disease. Part I, Vet. Rec. 70: 713-720; Part II, Vet. Rec., 70: 736-740.
12. McDIARMID, A. 1962. Diseases of free-living wild animals. FAO Agr. Studies No. 57, Rome. 10-11.
13. MEAD, C.J. 1968. Birds as vectors of the foot-and-mouth virus. In: *The Veterinary Annual*. Ed. by W.A. Pool. John Wright and Sons, Bristol.
14. MEANLY, B. and J.S. WEBB. 1965. Nationwide population estimates of blackbirds and starlings. Atlantic National, 20: 189-191.
15. PILCHARD, E.I. 1965. Experimental transmission of transmissible gastroenteritis virus by starlings. Am. J. vet. Res., 26: 1177-1179.
16. SCHWAB, R.G. 1967. Starling control research in California. Progress Report. Joint Report of the University of California Agr. Exp. Sta.; Bureau of Sports, Fisheries and Wildlife, U.S. Dept. of the Interior; and the Calif. Dept. of Agr.
17. Stockton Record newspaper, 1974. Millions of starlings peril Kentucky area. Stockton, California 79, 315: 2.
18. YATES, V.J. and L.T. MILLER. 1966. The isolation of avian tuberculosis from a starling. Bull. Wildl. Dis. Ass. 2: 84-85.

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