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Authors: JENSEN, WAYNE I., and COTTER, SALLY E.

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AN OUTBREAK OF ERYSIPELAS IN EARED GREBES *(Podiceps nigricollis)*

WAYNE I. JENSEN and SALLY E. COTTER, U.S. Fish and Wildlife Service,
Bear River Research Station, Brigham City, Utah 84302, USA

Abstract: An outbreak of erysipelas killed an estimated 5,000 aquatic birds on Great Salt Lake (Utah) in late November, 1975. Although several thousand ducks and gulls were using the lake, at least 99 percent of the victims were eared grebes. A hypothetical explanation for the selective mortality is offered.

INTRODUCTION

Erysipelas, a bacterial infection caused by *Erysipelothrix rhusiopathiae*, is best known as a disease of domestic animals, particularly swine and turkeys. However, reviews of the published literature on erysipelas in avian species by Levine[4] and Shuman[7] pointed out that it also has been reported in many wild birds, including aquatic species such as the mallard duck (*Anas platyrhynchos*) and herring gull (*Larus argentatus*). In their reviews, Levine and Shuman also include species of captive wild birds in which the disease has been reported. More recently Surkov *et al.*[9] isolated *E. rhusiopathiae* from the carrion crow (*Corvus corone*), European wigeon (*Anas penelope*), red-breasted merganser (*Mergus serrator*), and harlequin duck (*Histrionicus histrionicus*). MacDonald[5] reported erysipelas in single specimens of the sparrowhawk (*Accipiter nisus*), the hen harrier (*Circus cyaneus*), and the kittiwake (*Rissa tridactyla*). Munday[6] found agglutinins against *E. rhustiopathiae* in the sera of three of 27 forest ravens (*Corvus tasmanicus*), although he had no opportunity to attempt to isolate the bacterium.

E. rhusiopathiae has been reported in many species of mammals, including a wide variety of wild ones.[8] The infection in man (erysipeloid) is usually localized and self-limiting, but infrequently it may become generalized, with endocarditis as a complication.[1] Although Wood and Packer[10] found *E. rhusiopathiae* in manure and soil samples on swine-raising premises where the disease had not been seen for at least 5 years, they dispute the widely held belief that the organism exists for long periods of time as a saprophyte and suggest that, in their cases, it was frequently being shed by healthy, immune pigs. The length of time the site of an epizootic in wild birds might be expected to constitute a public health problem, therefore, is still in doubt.

This report describes an epizootic of erysipelas in the eared grebe (*Podiceps nigricollis*), which appears to have caused greater mortality than has been previously reported in wild birds.

CASE REPORT

History

In late November, 1975—the precise time is not known—a massive die-off of aquatic birds occurred on Great Salt Lake in northern Utah. A snowstorm on 26 and 27 November covered the carcasses, and they were not observed until 29 November, as the snow melted. The extent of the mortality became evident only on 10 December, when an aerial survey by Utah Division of Wildlife Resources personnel disclosed an estimated 5,000 dead birds along five km of the lake's southern shoreline. Although, according to observers, "thousands" of ducks were using the lake, at least 99% of the dead birds were eared grebes. The carcasses of a few herring gulls, green-winged teal (*Anas carolinensis*), shovelers (*Anas clypeata*), and common mergansers (*Mergus merganser americanus*) were also seen. One might suspect that

the disease was acute and the outbreak short-term, since there were no reports of sick birds having been seen, in spite of a considerable amount of hunting activity in the epizootic area. Hunting injury appeared to be the major cause of death among the ducks examined.

According to Wildlife Resources personnel, grebes and ducks appeared to have died on the water and were washed ashore, whereas the gulls died in the shallows along the shoreline. The gulls were observed feeding on carcasses, a likely source of infection for that species.

Necropsy

Of 11 frozen specimens submitted to the Bear River Research Station on 17 December, seven—four eared grebes, one shoveler, one herring gull, one common merganser—were examined both grossly and bacteriologically the following day. The remaining four grebes were retained for any virological, chemical, or other examination that might be indicated by preliminary observations.

Gross Examination

By 18 December, at least 3 weeks had passed since the estimated dates of the die-off. Although ambient temperatures had been generally low, the carcasses had not been constantly frozen. Gross pathologic changes may have been modified or obscured by the effects of autolysis and repeated freezing and thawing. Therefore, the descriptions that follow are undoubtedly incomplete.

All four grebes and one herring gull were in excellent physical condition, their subcutaneous fat layers ranging in thickness from 0.5 to 2 cm. Yellowish-orange livers were enlarged and friable; petechial hemorrhages in subcutaneous and pericardial fat were common. What appeared to be petechial hemorrhages on the pericardia and ecchymoses in the myocardia could have been the result, at least in part, of post-mortem autolysis.

The shoveler was thin almost to the point of emaciation. Several large blood clots were found in the pleural-peritoneal cavity. Although the merganser was in fair condition, as judged by the amount of body fat, it too had suffered extensive internal hemorrhages.

Bacterial examination

Liver, spleen, lung and heart blood from each bird were cultured individually, although the particular points of origin of the bacterium are probably not significant, because the semisolid consistency of some tissues increased the possibility of mechanical transfer of microorganisms from one to another.

A small portion of each tissue from the first bird of each species examined (grebe, gull, shoveler, merganser) was pressed to the surface of each of three Brain Heart Infusion Agar (BHIA) plates and three Cystine Heart Agar plates with Hemoglobin (CHHA), all Difco products.[1] The inocula so deposited were progressively thinned by means of four or five intersecting streaks across the plate with an inoculating loop. All plates were incubated at 37 C, one pair (BHIA and CHHA) aerobically, one pair anaerobically, and the third in a 10% carbon dioxide atmosphere. Tissues of the remaining three grebes were handled similarly, except that all plates were incubated aerobically. By the next day (about 20 h incubation), growth on all culture plates of tissues from four grebes and the one gull was thin, transparent, and confluent from the first impression to the fifth streak, indicating that the bacterium was present in the inocula in large numbers; and, judging from gram stains of growth taken from several sections of each plate, the cultures were pure. Colonies of many diverse species of contaminating bacteria were interspersed throughout the plate cultures of the shoveler and the merganser, but *E. rhusiopathiae* was the predominant organism.

[1] Reference to trade names does not imply endorsement of commercial products by the Federal Government.

The morphological and biological characteristics of *E. rhusiopathiae,* as demonstrated in this laboratory, are outlined below:

BHIA colonies were circular, entire, transparent, with bluish sheen by reflected light; pinpoint in size in 24 h to 1-2 mm in diameter at maturity when well isolated.

Cells gram-positive, short to long, sometimes curved, sometimes beaded, non-motile.

Characteristic "test tube brush" growth in gelatin stab (numerous radiating projections from line of stab). No liquefaction.

Acid but no gas from glucose, galactose, fructose, and lactose in CTA Medium (BBL Division of Becton, Dickinson and Co.)[1] No acid from rhamnose, inulin, saccharose, mannitol, raffinose, trehalose, sorbital, melibiose, and xylose.

Hydrogen sulfide produced in Kligler Iron Agar and Triple Sugar Iron Agar. Indole not produced, urea not hydolyzed, citrate not utilized, nitrate not reduced.

Physiological saline extracts of the livers of four birds—two grebes, one herring gull, one shoveler—caused the death of laboratory mice in 36 to 48 h after intraperitoneal (ip) injection, and the bacterium was isolated from the mouse livers and heart blood in each case. Two mallard ducks, however, did not show overt signs of infection with our strain of *E. rhusiopathiae* when administered either by ip injection or by way of traumatized conjunctivae.

Additional bird carcasses, collected on the southern shore of Great Salt Lake on 7 January, 3 February and 3 March 1976, were stored in outdoor cabinets where the air temperature is similar to that on the lake shore. One or two specimens were examined each week up to 23 April as a means of judging the viability of the bacterium in wild bird carcasses. The findings in 15 birds (in addition to the original seven) are summarized here:

Postmortem decomposition became progressively obvious with time, both by gross appearance and by bacteriological examinations. The internal organs in some specimens had lost their identity and, in general, the species and numbers of postmortem contaminants increased each week.

E. rhusiopathiae was isolated from all 10 of the grebes included in the more recent samplings, one of which had been dead an estimated 18 weeks. It was usually present in large numbers in all tissues examined; in only one instance was it necessary to use mouse inoculation to separate the pathogen from more abundant contaminants.

Even though one herring gull was badly decomposed. *E. rhusiopathiae* was the predominant organism on the culture plates and was easily isolated. No evidence of erysipelas could be found in three shovelers, two of which showed evidence of traumatic injury (extensive internal hemorrhages). The third carcass was badly decomposed and no meaningful judgment could be made. A green-winged teal also had suffered internal hemorrhages and no known pathogenic bacterium could be isolated from its tissues.

In summary, *E. rhusiopathiae* was isolated from all of 14 grebes, both herring gulls, one of four shovelers, and one common merganser collected between 13 December 1975, and 3 March 1976. It was not found in the only green-winged teal examined.

DISCUSSION

Although the source of the infective agent responsible for this outbreak was not ascertained, healthy swine may serve as carriers of *E. rhusiopathiae,*[2,3] and swine are raised on farms within a few miles of Great Salt Lake. Shuman[7] believes that carrier birds or mammals may account for some outbreaks in birds.

It is possible, of course, that a few or many of the grebes themselves were carriers. Defense of such a hypothesis would require identifying factors that may have precipitated the outbreak, unusual stresses for example that could have lowered

the birds' resistance to the potential pathogens they carried. There is little doubt that the grebes were in the course of their southward migration and that they had stopped on the lake to rest and feed upon its abundant brine shrimp (*Artemia salina*). One could speculate that, being weak fliers and just having completed a long, strenuous flight, they were more severely stressed by the late November snowstorm than were the other species of birds using the lake.

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LITERATURE CITED

1. BEESON, P. B. and W. McDERMOTT. *Textbook of Medicine,* 13th ed. W. B. Saunders Co., 1971. Philadelphia.
2. BLOOD, D. C. and J. A. HENDERSON. *Veterinary Medicine,* 4th ed. Williams and Wilkins Co., 1974. Baltimore.
3. BRUNER, D. W. and J. H. GILLESPIE. *Hagan's Infectious Diseases of Domestic Animals,* 6th ed. Cornell Univ. Press, 1973. Ithaca.
4. LEVINE, N. D. Erysipelas. In H. E. Biester and L. H. Schwarte, eds. *Diseases of Poultry,* 5th ed. Iowa State Univ. Press, 1965, Ames.
5. MACDONALD, J. W. 1968. Listeriosis and erysipelas in wild birds. Bird Study 15: 37-38.
6. MUNDAY, B. L. 1972. A serological study of some infectious diseases of Tasmanian wildlife. J. Wildl. Dis. 8: 169-175.
7. SHUMAN, R. D. Erysipelothrix. In J. W. Davis, R. C. Anderson, L. Karstad and D. O. Trainer, eds. *Infections and Parasitic Diseases of Wild Birds.* Iowa State Univ. Press, 1972. Ames.
8. ————. Erysipelas. In J. W. Davis, L. Karstad and D. O. Trainer, eds., *Infectious Diseases of Wild Mammals.* Iowa State Univ. Press, 1970. Ames.
9. SURKOV, V. S., N. S. TIMOFEEVA and N. G. SUCHKOVA. 1972. Natural sources of erysipeloid in Sakhalin. Zh. Mikrobiol. Epidemiol. Immunobiol. 49: 3-5.
10. WOOD, R. L. and R. A. PACKER. 1972. Isolation of *Erysipelothrix rhusiopathiae* from soil and manure of swine-raising premises. Am. J. vet. Res. 33: 1611-1620.

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