# Plague in a Free-ranging Mule Deer from Wyoming

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TABLE 1. Occurrence of neutralizing activity specific for the striped bass isolate of infectious pancreatic necrosis virus in striped bass from the Chesapeake Bay, Maryland.

| Survey date | Location | Year-class | No. positive/no. tested* |
|---|---|---|---|
| Dec. 1984 | Choptank River | 1982–1983 | 9/49 (18%) |
| Feb. 1985 | Upper Bay | 1982–1983 | 6/94 (6%) |
| July 1985 | Upper Bay | 1985 | 0/5 |
| Aug. 1985 | Choptank River | 1985 | 1/45 (2%) |
| Aug. 1985 | Upper Bay | 1985 | 0/3 |
| Sept. 1985 | Upper Bay | 1985 | 0/6 |
| Sept. 1985 | Upper Bay | 1984 | 0/20 |

* Number of striped bass blood samples (diluted 1:100) that neutralized more than 50% of total plaques of the striped bass isolate of IPNV, but did not neutralize the European IPNV isolate (IPNV-Ab) per number of samples tested.

(McAllister et al., 1984, Helgol. Meeresunters. 37: 317–328), this is the first report of IPNV-neutralizing activity in a wild, nonsalmonid species of fish.

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## Plague in a Free-ranging Mule Deer from Wyoming

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Plague, a bacterial disease caused by *Yersinia pestis*, occurs in many areas of the western United States and other regions of the world. It commonly involves rodents (e.g., sciurid ground squirrels) and associated carnivores or scavenger species preying upon affected rodent populations. Occasionally humans become infected, either by the bite of infective rodent fleas or by direct handling of infective animals.

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Reports of plague in big game or domestic animals, however, have been limited. Federov (1960, Bull. W.H.O. 23: 275–281) summarized early reports of natural and experimental plague infections in camels (*Camelus bactrianus* and *C. dromedarius*) in Russia. Two outbreaks of human plague in Libyan villages in 1972 and 1977 were traced to the preparation and consumption of meat from infected camels (presumably *C. dromedarius*) (Bytchenko, 1976, cited by B. Velimirovic, 1979, *In* CRC Handbook Series in

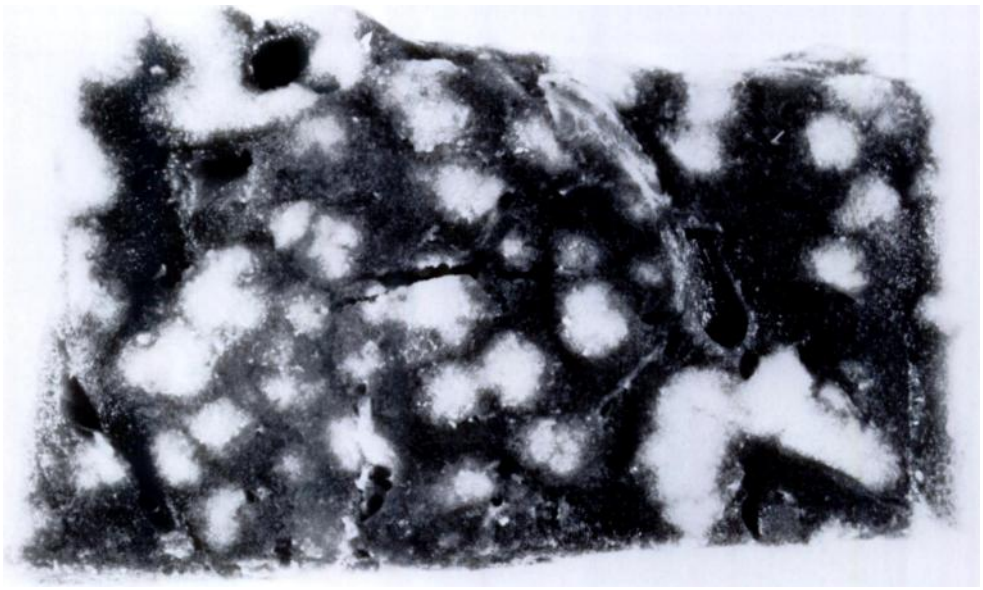

FIGURE 1. Formalin-fixed section of lung from a mule deer with plague containing numerous white areas of necrosis and neutrophilic infiltration.

Zoonoses, Section A: Bacterial, Rickettsial and Mycotic Diseases, Vol. 1, Steele (ed.), CRC Press, Cleveland, Ohio, pp. 560–596). Gordon et al. (1979, Infect. Immun. 26: 767–769) found specific serologic evidence of plague infection in 26 of 391 African buffaloes (*Syncerus caffer*) and one of 330 African elephants (*Loxondonta africana*), but in none of 16 Burchell's zebra (*Equus burchelli*) or five sable antelope (*Hippotragus niger*) at Wankie National Park in Zimbabwe 1 yr after a plague epidemic in the area.

Presence of plague has been recognized in Wyoming since 1936 when *Y. pestis* was isolated from fleas collected from ground squirrels [*Spermophilus* (*Citellus*) *armatus*] in Yellowstone National Park (Quan, 1982, *In* Diseases of Wildlife in Wyoming, Thorne et al. (eds.), Wyoming Game and Fish Department, Cheyenne, p. 68). A variety of rodents, lagomorphs, carnivores, arthropods, and several humans have shown evidence of infection with *Y. pestis* in Wyoming (Quan, 1982, op. cit.) and outbreaks of plague are documented within the state almost yearly. To our knowledge, this report describes the first known case of plague in a cervid, a free-ranging mule deer (*Odocoileus hemionus*) from Wyoming.

In April 1980, a female mule deer fawn was observed in a weakened condition near a cattle feed lot in Niobrara County in eastern Wyoming. Clinical signs included labored breathing, ataxia, and hypersalivation. The fawn was manually captured and died while being restrained. It was transported to the Wyoming Game and Fish Department's Sybille Wildlife Research Unit for necropsy.

The fawn was judged to be in fair body condition based on distribution of body fat and had not yet begun to shed the winter hair coat. Fibrinous adhesions were present between the visceral and parietal pleura. The lungs were collapsed slightly and studded with numerous firm, raised,

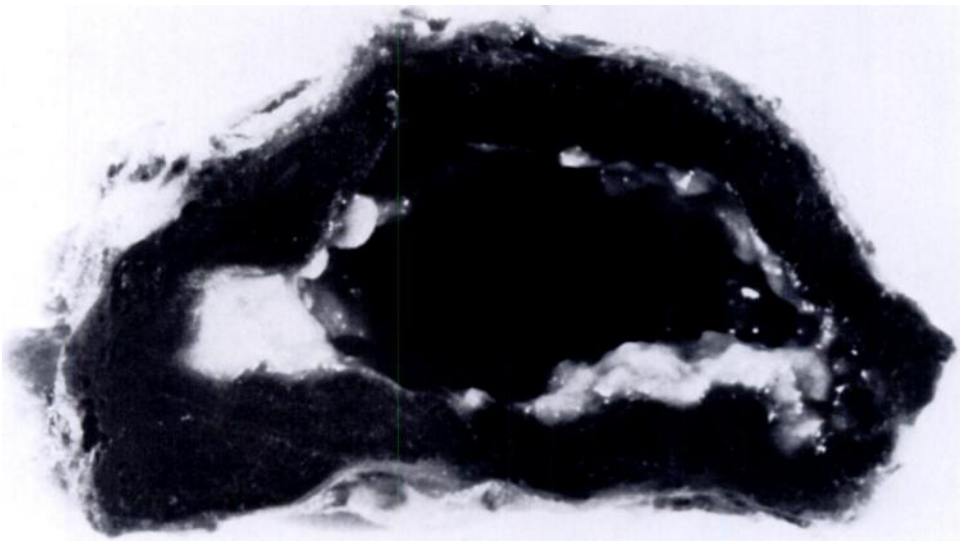

FIGURE 2. Cross section of a formalin-fixed lymph node from the deer. Note large cavity resulting from necrosis in the medullary region.

white 1–3 mm diameter foci on pleural and cut surfaces (Fig. 1) which were most numerous in the diaphragmatic lobes. The trachea was filled with white foam. Mandibular, pharyngeal, cervical, mediastinal, and tracheobronchial lymph nodes were enlarged, firm, and dark red. Variable-sized necrotic areas, containing thick yellow-white fluid, were present in some lymph nodes (Fig. 2). Small focal hemorrhages occurred in adrenal cortex and medulla and in intercostal musculature. Skeletal muscles of the legs were pale.

Microscopically, the lungs were characterized by widespread foci of necrosis containing large, pale basophilic bacterial colonies surrounded by an infiltrate of neutrophils. Eosinophilic fluid and fibrin were present in alveoli and airways. Extensive necrosis was seen in the medulla of mandibular and pharyngeal lymph nodes and bacterial colonies were numerous. A severe degenerative myopathy was characterized by variation in size and staining of muscle fibers, granularity and basophilia of fibers, proliferation of sarcolemmal nuclei, and a mild mononuclear inflammatory cell infiltrate.

Wayson's stained impression smears of the lung demonstrated numerous bipolar bacilli. Culture of lung and lymph node using a commercial identification system for bacteria (API 20E, Analytab Products, Inc., Plainview, New York 11803, USA) indicated the isolate was *Y. pestis*.

Fluorescent antibody tests for *Y. pestis* and *Francisella tularensis* were performed according to methods of Winter and Moody (1959, J. Infect. Dis. 104: 281–287) and Moody and Winter (1959, J. Infect. Dis. 104: 288–294) using reagents prepared at the Laboratory of Plague Branch, Division of Vector-Borne Viral Diseases, Centers for Disease Control, Fort Collins, Colorado. The fluorescent antibody test for tularemia was negative. The

test for plague infection revealed bacterial cells which fluoresced specifically and were of size and morphology consistent with *Y. pestis*.

Portions of lung and lymph node were triturated with sterile sand and suspended in sterile physiological saline. Laboratory mice (NIH General Purpose Strain, from a specific pathogen-free mouse colony) were inoculated subcutaneously with 0.5 ml of tissue suspension. Mice died by the third day postinoculation.

Spleen and liver tissues of dead mice were examined by the fluorescent antibody test and were cultured in duplicate for recovery of *Y. pestis*, *F. tularensis*, and other agents using blood agar and cystine heart agar supplemented with 8% sheep blood. Inoculated media were incubated at 28 C and 37 C in a humid chamber and examined daily for growth. Tissues were positive for *Y. pestis* by the fluorescent antibody test and culture yielded colonies consistent with this agent.

The characteristics on which the identification of *Y. pestis* was based included: gram negative rod or coccobacillus; positive, specific fluorescence with high-titered anti-*Y. pestis* rabbit serum conjugated with fluorescein isothiocyanate; lysis by *Y. pestis*-specific bacteriophage at both 25 C and 37 C; colonial size and morphology; pathogenicity for laboratory mice; and biochemical reactions.

The possibility of plague among ungulate populations has not been investigated thoroughly in this country and little is known of the potential for epizootics among herds in plague areas. Ungulate habitat is shared by many rodent reservoirs with ample opportunity for exchange of ectoparasites. Routes of infection other than by bites of infective ectoparasites are only remotely plausible, but would include consumption of forage or water contaminated by feces, urine, or blood of infected rodents or by infected fleas. Russian studies summarized by Federov (1960, op. cit.) indicated camels (and presumably other herbivores) were heterogenous in their response to the same challenge of *Y. pestis* and that susceptibility varied depending on route of inoculation. For example, all animals given an aerosol challenge died of typical primary plague pneumonia; subcutaneous inoculation with "massive doses" of *Y. pestis* killed half the experimental camels; and camels infected orally developed mild, non-fatal forms of the disease.

Not known in the present case are the predisposing factors which allowed development of fatal plague in the deer, e.g., host condition, source of infection, route of infection, numbers of organisms gaining entry, or the number of challenges inflicted upon the animal. Plague activity had not been documented in Niobrara County, Wyoming prior to this case. Possibly this deer's fatal infection with plague represented an individual response and is not indicative of a general susceptibility of deer to plague. If cervids were highly susceptible, other cases should have been recognized because *Y. pestis* is easily recovered, cultivated, and identified by standard bacteriological procedures. Also, human infections following contact with the game species during hunting seasons and by wildlife specialists in all seasons should be more prevalent were plague a common occurrence in these animals.

A valuable lesson from this case is that prejudgments as to the etiology in any sick or dead animal may be erroneous, and potentially dangerous. *Yersinia pestis* was not among the pathogenic organisms suspected of causing the illness and death of the deer. The considerable exposure experienced by the rancher and game warden in capturing, restraining, and transporting the deer with pneumonic plague did not result in human infection. Standard safety procedures followed during necropsy (gloves, disinfecting the area) and during preliminary microbiological studies served

to protect exposed personnel and point to the importance of these routines.

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# Ostertagiosis in a White-tailed Deer due to *Ostertagia ostertagi*

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A debilitated yearling male white-tailed deer (*Odocoileus virginianus*) was found in a cattle pasture in Wilkes County, Georgia, on 2 April 1985 and submitted to the Southeastern Cooperative Wildlife Disease Study for examination. The deer was emaciated (20 kg) and covered by thousands of lonestar ticks (*Amblyomma americanum*), especially on the ears and around the anus. The latter was swollen and contained numerous purulent fistulous tracts. The animal could not stand, and neither deep pain reflexes nor a panniculus response could be elicited. Tick paralysis was suspected, the animal was lightly tranquilized (Rompun), and a large number of ticks were individually removed with forceps. The deer was then dipped with an organophosphate pesticide (Dermaton H) to kill the remaining ticks and given an intramuscular injection of penicillin. The deer died during the night and was necropsied the following morning.

At necropsy, there was widespread individual to confluent nodular thickening of the abomasal mucosa which spared the pyloric antrum (Fig. 1). The margins of the mucosal folds were most severely involved and had scattered petechial and ecchymotic hemorrhages. Generalized enlargement of lymph nodes, splenic lymphoid hyperplasia, serous atrophy of fat, pale and swollen kidneys, and a few small, poorly circumscribed, pale foci in the heart were present. On a blood smear, many monocytes contained a granular rickettsial-type intracytoplasmic inclusion body, as well as a variety of bacilli and cocci. Mononuclear cells in lymph node smears contained similar rickettsial-type inclusion bodies and bacteria. Tentative diagnoses of ostertagiosis, rickettsiosis, and bacteremia secondary to tick infestation were made.

A dilution count (Eve and Kellogg, 1977, J. Wildl. Manage. 41: 169–177) was performed to estimate the total number of nematodes in the abomasum. A total of 3,660 nematodes was estimated, composed of approximately 2,620 adult and 1,040 larval forms. Of the adult total, ratios of identifiable male worms showed 68.4% (1,793) of the nematodes recovered to be the medium stomach worm, *Ostertagia ostertagi*. The remaining parasites were two other medium stomach worms, *Ostertagia dikmansi* (2.6%), and *Apteragia odocoilei* (2.6%), the large stomach worm, *Haemonchus contortus* (7.9%), and three small stomach worms, *Trichostrongylus axei* (10.5%), *T. askivali* (5.3%), and *T. dosteri* (2.6%). Morphologically, it

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