(33%) originating from the Alaska Peninsula and nine samples (23%) from the Susitna area. Positive titers ranged from 1:16 to 1:128. Six samples (6%) were seropositive to IBRV, with four (10%) being from the Susitna area and two (17%) from the Alaska Peninsula. All positive titers were at the 1:16 dilution. Antibodies to IBRV in moose have been reported previously for 14% of 14 moose sampled in Alberta (Zarnke and Yuill, 1981, J. Wildl. Dis. 17: 453–461) although none were found in a previous study (Thorsen and Henderson, 1971, J. Wildl. Dis. 7: 93–95). Unpublished studies on serologic reactivity to IBRV and BVDV apparently have shown seroreactive moose in Alaska (Dieterich, 1981, op. cit.) although evaluation of 73 Alaskan Dall's sheep (*Ovis dalli*) failed to detect serologic reactivity to either virus (Foreyt et al., 1983, J. Wildl. Dis. 19: 136–139) nor did an evaluation of 39 moose from Alaska result in the detection of antibodies to BVDV or IBRV (Zarnke et al., 1983, op. cit.). The detection of serologic reactivity to these two viruses in the present study indicates that contact with domestic cattle or other seropositive wild ruminants may have occurred. Further studies appear warranted to determine the role of moose in the epidemiology of these infectious diseases and the significance of these findings to moose health.

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Isolation of a Poxvirus from a House Finch, *Carpodacus mexicanus* (Müller)

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Avian pox has been reported in many bird species (Kirmse, 1967, J. Wildl. Dis. 3: 14–20). On two occasions pox lesions have been observed on the house finch (*Carpodacus mexicanus* Müller), but virus was not isolated. Warner (1969, Condor 70: 101–120) first observed pox lesions on house finches introduced to Hawaii. He reported that nearly half of the house finches trapped had lesions at the bend of the wing, lores, and/or tarsal joint. None of the lesions were severe and it was suggested that the house finch was resistant to the virus. Power and Human (1976, Condor 78: 262–263) described an outbreak of disease among house finches frequenting bird feeders in the Santa Barbara, California area during the winter of 1971–1973. Of the total number of house finches captured, 17% (7/42) died with lesions. The authors noted that some of these birds had lesions severe enough to have caused mortality in the wild. In one of the captive birds, a minor lesion near one eye progressed in 3 wk to the point of completely closing both eyes. We report here the first isolation of poxvirus from the house finch.

In February 1983, two house finches found dead at a bird feeder in Boise, Idaho were submitted to the U.S. Fish and Wildlife Service, National Wildlife Health Laboratory, Madison, Wisconsin. Lesions were present near the beak and on the legs of both birds (Fig. 1). In both cases, the lesions on the beak were extensive enough to obstruct vision.

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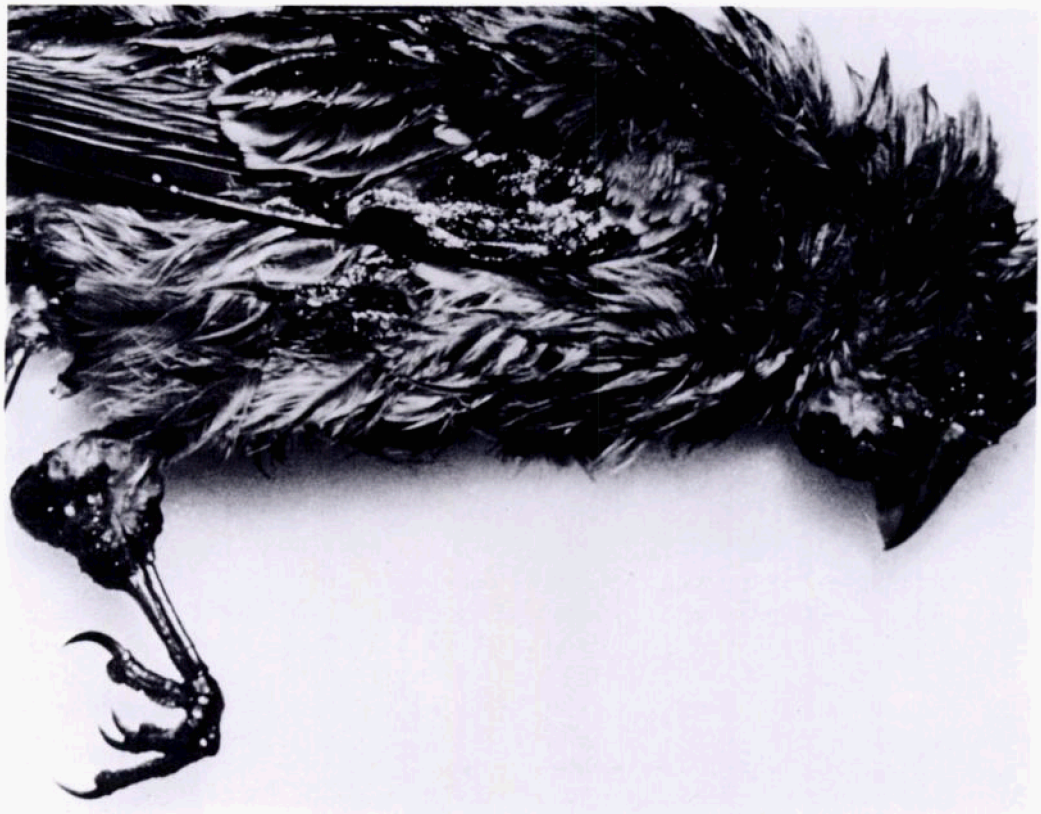

FIGURE 1. Location of pox lesions on a house finch.

The lesions, consisting of excised tissue, were pooled, diluted 1:10 in Hanks' balanced salt solution (BSS), ground in a Waring blender, centrifuged at 200 *g* for 30 min and passed through a 0.45-μm Millipore filter. This suspension was inoculated onto the chorioallantoic membrane (CAM) of embryonated chicken eggs (Tripathy and Hanson, 1975, *In* Isolation and Identification of Avian Pathogens, Hitchner et al. (eds.), Arnold Printing Corp., Ithaca, New York, pp. 282–290), and muscovy duck embryo fibroblast (MSDE-F) cell culture prepared by standard methods (Rovozzo and Burke, 1973, A Manual of Basic Virological Techniques, Prentice-Hall, Inc., Englewood Cliffs, New Jersey, pp. 41–43).

Five days postinoculation, all of the CAM's were noticeably thickened at the site of inoculation. The 50% infectious dose (ID_{50}) of the original suspension titered on CAM's was $10^{6.5}$ ID_{50}/ml. Discrete pocks were formed on the CAM of eggs inocu-

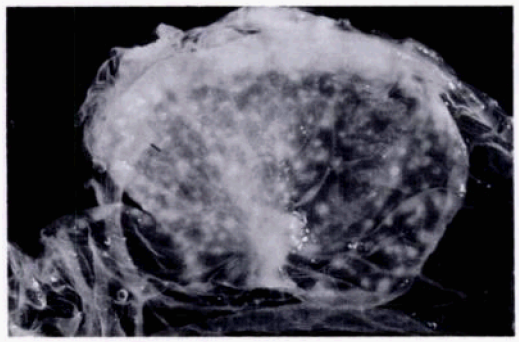

FIGURE 2. Pox lesions on the chorioallantoic membrane of an embryonated chicken egg inoculated with a suspension prepared from house finch pox lesions.

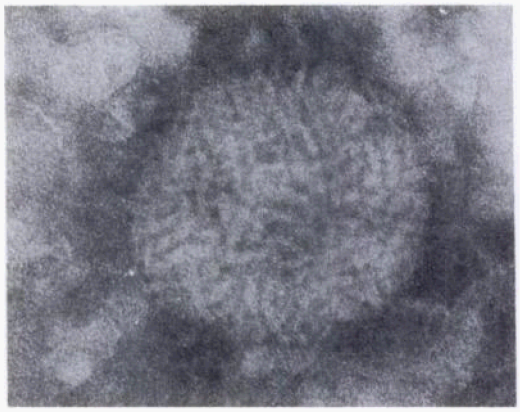

FIGURE 3. Electron micrograph of house finch poxvirus grown on the chorioallantoic membrane of an embryonated chicken egg. Phosphotungstic acid, ×140,000.

lated with dilutions near the ID_{50} endpoint (Fig. 2). A pool of the infected CAM's was diluted 1:10 in Hanks' BSS, ground up in a Waring blender, and centrifuged at 35,000 *g* for 30 min. The pellet was resuspended in distilled water, negative stained with phosphotungstic acid, and viewed with a H500 Hitachi electron microscope. Numerous poxvirus virions (300 × 250 nm) were seen (Fig. 3).

No viral cytopathic effect (CPE) was noted in cell culture after two blind passages of the original pox suspension in MSDE-F. However, CPE did occur in MSDE-F 3 days postinoculation with a suspension prepared from infected CAM's. The cytopathic effect consisted of rounding of cells followed by degeneration resulting in plaques. The tissue culture ID_{50} titer was $10^{6.04}$/ml after passage in CAM and three passages in MSDE-F.

The outbreaks of poxvirus disease among house finches in Boise, Idaho and Santa Barbara, California were noted in bird populations utilizing feeders. Feeders are often placed to facilitate bird observation by humans, making it easier to detect birds with obvious lesions. Because feeders concentrate birds, there is a greater possibility of disease transmission between birds. Parts of the feeder may become contaminated with poxvirus and transmission could also occur after the infected bird has left. The possibility of transmission is further increased if the feeder is constructed of materials that could cause trauma to the beak, legs, or feet while feeding. The resulting minor cuts are sufficient to admit pathogens (Karstad, 1971, *In* Infectious and Parasitic Diseases of Wild Birds, Davis et al. (eds.), Iowa State Univ. Press, Ames, Iowa, pp. 34–41).

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