

FIRST CASE OF *MYCOPLASMA GALLISEPTICUM* INFECTION IN THE WESTERN RANGE OF THE HOUSE FINCH (*CARPODACUS MEXICANUS*)

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ABSTRACT.—We report the first case of mycoplasmosis in the western range of the House Finch (*Carpodacus mexicanus*). This disease originated in the eastern United States and has been previously documented only in eastern introduced House Finch populations where it reached epizootic proportions causing extensive and widespread mortality. Documentation of this disease in western Montana suggests that previously disjunct eastern and western populations of House Finches are now mixing in the northern part of their range. More importantly, as native House Finches are highly susceptible to this novel pathogen, western populations may now be at risk of high mortality, similar to that experienced by non-native eastern populations. Close monitoring of this disease in the western part of the House Finch range will provide important insight into the dynamics of the emerging disease and evolution of resistance to the pathogen. Received 10 May 2002, accepted 5 February 2003.

RESUMEN.—Reportamos el primer caso de mycoplasmosis en el rango de distribución oeste de *Carpodacus mexicanus*. Esta enfermedad se originó en el este de los Estados Unidos y ha sido documentada previamente sólo en poblaciones de *C. mexicanus* introducidas en el este, donde alcanzó proporciones epizooticas causando una mortalidad amplia y difundida. El registro de esta enfermedad en el oeste de Montana sugiere que poblaciones del este y del oeste de *C. mexicanus* previamente disyuntas se están mezclando actualmente en la parte norte de su rango de distribución. Aún más importante, debido a que individuos nativos de la especie *C. mexicanus* son altamente susceptibles a este nuevo patógeno, las poblaciones del oeste pueden tener un alto riesgo de mortalidad similar al observado en poblaciones introducidas del este. Un monitoreo cuidadoso de esta enfermedad en la parte oeste del rango de distribución de *C. mexicanus* proporcionará información importante sobre las dinámicas de la enfermedad emergente y evolución de la resistencia al patógeno.

MYCOPLASMA GALLISEPTICUM INFECTION, a common disease of poultry, was first diagnosed in the House Finch (*Carpodacus mexicanus*) in January 1994 in Maryland. This disease spread rapidly in House Finch populations throughout the eastern United States, and in less than a year cases were described in populations from New York to North Carolina (Fischer et al. 1997). By 1997 the disease had reached epizootic proportions, having spread across the entire eastern range of the House Finch, killing an estimated 225 million birds (Nolan et al. 1998).

Eastern House Finch populations are derived from ~80 individuals released in Long Island, New York, in the early 1940s. Around 1960, that population, as well as native populations

in southwestern United States, began to expand their range (Hill 1993). However, during the time of the *Mycoplasma gallisepticum* infection epizootic in the east, the eastern and western populations of the House Finch remained isolated from each other. To this date, there have been no confirmed cases of mycoplasmosis crossing into the western range of the House Finch.

Birds infected with mycoplasmosis (an upper respiratory tract disease) suffer from tearing, redness, and swelling of the conjunctival tissues and nasal discharge—clinical signs that are easily observed in the field (Fig. 1). On 8 April 2002, we observed a male House Finch with a moderately swollen conjunctiva at a feeding station in Missoula, Montana (46°52'N, 114°00'W). That feeding station was located within a population of resident, individually marked House Finches (see Badyaev and Martin 2000). The population has been monitored since 1994, and

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Fig. 1. Swelling of conjunctival tissues observed in two male House Finches from Missoula, Montana that tested positive for mycoplasmosis in March–May 2002.

that was the first symptomatic bird out of the ~4,900 finches captured in that and other areas of Missoula, Montana. Within 30 min of seeing the symptomatic male, we captured it using a corridor of mist nets set around the feeding station.

A blood sample from the male was obtained and was scored for hematocrit. Plasma was saved for analyses. Using a microtip swab (Becton Dickinson and Co., Sparks, Maryland), we took samples from the swollen conjunctiva and the choanal cleft for PCR analysis. In addition, we weighed and photographed the male before euthanizing with carbon monoxide. Both the plasma sample and the carcass were stored on ice and shipped overnight for laboratory testing.

Two independent researchers tested the serum for antibodies of *Mycoplasma gallisepticum* infection with a commercial serum plate agglutination (SPA) assay (Luttrell 1996) (Intervet Millsboro, Delaware). After two minutes, the agglutination was scored on a scale of 0–4 with 2 considered positive. DNA was extracted from swabs by boiling them in 100 μ L of vaccine water for 10 min followed by a 10-min freezing. PCR amplification of a 185-bp fragment using *Mycoplasma gallisepticum* specific primers (Life Technologies Gaithersburg, Maryland) was performed both in our laboratory and in the C. S. Roberts Veterinary Diagnostic Laboratory to assure independent testing (case no. 02D06662). Swabs were placed into an SP4 broth tube prewarmed to 37°C. A 1:10 blind passage was made 24 hr following initial culture. The broth culture was incubated at 37°C for three week until a phenol-red-indicated color change occurred, at which time the culture was tested for *Mycoplasma gallisepticum* infection by PCR.

SPA of the serum from the House Finch scored as a 3 in two independent tests confirming the presence of *Mycoplasma gallisepticum* antibodies. Extractions from the swabs of the conjunctiva and choanal cleft tested positive for *Mycoplasma gallisepticum* by PCR at both testing facilities. After six weeks the culture changed from red to yellow, indicating growth of the Mycoplasma. PCR confirmed that the growth was *Mycoplasma gallisepticum*. Tests in both testing facilities confirmed the male House Finch was positive for *Mycoplasma gallisepticum*. In addition, the male's weight (18.4 g) and hematocrit (54%) were below the average (20 g and 59%) compared to other males ($n = 15$) caught at the study site the same day suggesting that the male was in poor overall condition. In the two months after initial discovery of *Mycoplasma gallisepticum* infection, two more male House Finches with mycoplasmosis symptoms were captured at the Missoula site on 20 and 22 May 2002 and subsequently tested positive for *Mycoplasma gallisepticum* infection using the PCR analysis. Morphological measurements of these two males were within normal range.

Detecting three cases of mycoplasmosis in northwest Montana suggests divergent House Finch populations have now come in contact in the northern part of their range. Moreover, western House Finches, including those from the Montana populations, are highly susceptible to *Mycoplasma gallisepticum* (Farmer et al. 2002) and consequently, this highly contagious disease might cause a significant decline in native populations of the House Finch. Careful monitoring of the emergence of this disease in the previously unexposed western House Finch populations can provide insight into the ecology and evolution of host–parasite interactions.

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