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Abstract
In 1994, an endemic poultry pathogen, *Mycoplasma gallisepticum* (MG), was identified as the causative agent of a novel disease in house finches (*Haemorhous mexicanus*). After an initial outbreak in Maryland, MG spread rapidly throughout eastern North American populations of house finches. Subsequently, MG spread slowly through the northern interior of North America and then into the Pacific Northwest, finally reaching California in 2006. Until 2009, there were no reports of MG in the southwestern United States east of California. In August 2011, after reports of house finches displaying conjunctivitis characteristic of MG infection in Arizona, we trapped house finches at bird feeders in central Arizona (Tempe) and southern Arizona (Tucson and Green Valley) to assay for MG infection. Upon capture, we noted whether birds exhibited conjunctivitis, and we collected choanal swabs to test for the presence of MG DNA using PCR. We detected MG in finches captured from Green Valley (in ~12% of birds captured), but not in finches from Tucson or Tempe. Based on resampling of house finches at these sites in July 2014, we suggest that central Arizona finches likely remain unexposed to MG. We also suggest that low urban connectivity between arid habitats of southern and central Arizona or a reduction in the prevalence of MG after its initial arrival in Arizona may be limiting the spread of MG from south to north in Arizona. In addition, the observed conjunctivitis-like signs in house finches that were negative for MG by PCR may be caused primarily by avian pox virus.

Keywords
Mycoplasma gallisepticum, house finch, Haemorhous mexicanus, conjunctivitis

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Detection of *Mycoplasma gallisepticum* in House Finches (*Haemorhous mexicanus*) from Arizona

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SUMMARY. In 1994, an endemic poultry pathogen, *Mycoplasma gallisepticum* (MG), was identified as the causative agent of a novel disease in house finches (*Haemorhous mexicanus*). After an initial outbreak in Maryland, MG spread rapidly throughout eastern North American populations of house finches. Subsequently, MG spread slowly through the northern interior of North America and then into the Pacific Northwest, finally reaching California in 2006. Until 2009, there were no reports of MG in the southwestern United States east of California. In August 2011, after reports of house finches displaying conjunctivitis characteristic of MG infection in Arizona, we trapped house finches at bird feeders in central Arizona (Tempe) and southern Arizona (Tucson and Green Valley) to assay for MG infection. Upon capture, we noted whether birds exhibited conjunctivitis, and we collected choanal swabs to test for the presence of MG DNA using PCR. We detected MG in finches captured from Green Valley (in ~12% of birds captured), but not in finches from Tucson or Tempe. Based on resampling of house finches at these sites in July 2014, we suggest that central Arizona finches likely remain unexposed to MG. We also suggest that low urban connectivity between arid habitats of southern and central Arizona or a reduction in the prevalence of MG after its initial arrival in Arizona may be limiting the spread of MG from south to north in Arizona. In addition, the observed conjunctivitis-like signs in house finches that were negative for MG by PCR may be caused primarily by avian pox virus.

RESUMEN. Detección de *Mycoplasma gallisepticum* en pinzones mexicanos (*Haemorhous mexicanus*) de Arizona.

En 1994 se identificó *Mycoplasma gallisepticum* (MG), que es un patógeno endémico en la avicultura, como el agente causante de una nueva enfermedad en pinzones mexicanos (*Haemorhous mexicanus*). Después de un brote inicial en Maryland, *M. gallisepticum* se extendió rápidamente a lo largo de las poblaciones de pinzones domésticos en el este de América del Norte. Posteriormente, *M. gallisepticum* se extendió lentamente por el interior del norte de América del Norte y luego en el noroeste del Pacífico, llegando finalmente a California en el año 2006. Durante 2009, no hubo informes de *M. gallisepticum* en el suroeste de los Estados Unidos y la parte este de California. En agosto del 2011, después de informes de pinzones que mostraron conjuntivitis característica de la infección por *M. gallisepticum* en Arizona, se atraparon pinzones mexicanos en comederos de aves en el centro de Arizona (Tempe) y en el sur de del mismo estado (Tucson y Green Valley) para evaluar la infección por *M. gallisepticum*. Durante la captura, se observaron las aves para detectar si exhibían conjuntivitis, se recolectaron hisopos de coanas para evaluar la presencia de ADN de *M. gallisepticum* mediante PCR. Se detectó M. gallisepticum en pinzones capturados en Green Valley (en aproximadamente el 12% de las aves capturadas), pero no en los pinzones capturados en Tucson o en Tempe. Con base en un remuestreo de pinzones mexicanos en estos sitios en julio del 2014, se sugirió que los pinzones del centro de Arizona probablemente no estaban expuestos a *M. gallisepticum*. También se sugirió que la baja conexión urbana entre los hábitats áridos del sur y del centro de Arizona o una reducción en la prevalencia de M. gallisepticum después de su llegada inicial a Arizona puede estar limitando la propagación de *M. gallisepticum* de sur a norte en Arizona. Además, los signos similares a la conjuntivitis observados en los pinzones que fueron negativos para *M. gallisepticum* mediante PCR pueden ser causados principalmente por poxvirus aviar.

Key words: *Mycoplasma gallisepticum*, house finch, *Haemorhous mexicanus*, conjunctivitis

Abbreviations: MG = *Mycoplasma gallisepticum*

The colonization of a novel host by a pathogen offers the opportunity to study the conditions under which host shifts occur, the mode and tempo of transmission through the novel host population, and changes in host-pathogen dynamics through time (2,6,10,21). For such studies to be most revealing, accurate surveys of pathogen prevalence throughout the range of the host, particularly near the leading edge of pathogen spread, are critical.

One of the most extensively documented host shifts is that of the bacterial pathogen *Mycoplasma gallisepticum* (MG) into North American house finches (*Haemorhous mexicanus*), with genetic analyses indicating a single host shift of MG from poultry (8,23). First detected in Maryland in 1994, MG spread rapidly through eastern North American house finch populations (15,23). This epizootic was unusually well documented because of the visible conjunctivitis caused by MG as well as via the active disease monitoring by biologists and through the House Finch Disease Survey organized by the Cornell University Laboratory of Ornithology (12,15,17,25,32). Within a year of the outbreak, MG had spread beyond the Mid-Atlantic states and reached all of New England and as far south as Georgia. House finches with conjunctivitis were reported in all states east of the Rocky Mountains by 1997 (12,15), and the entire eastern North American house finch population was reduced to about half of its pre-epizootic size (21,27).

In contrast to its rapid epizootic spread, MG has spread more slowly through western North America. In addition, western MG

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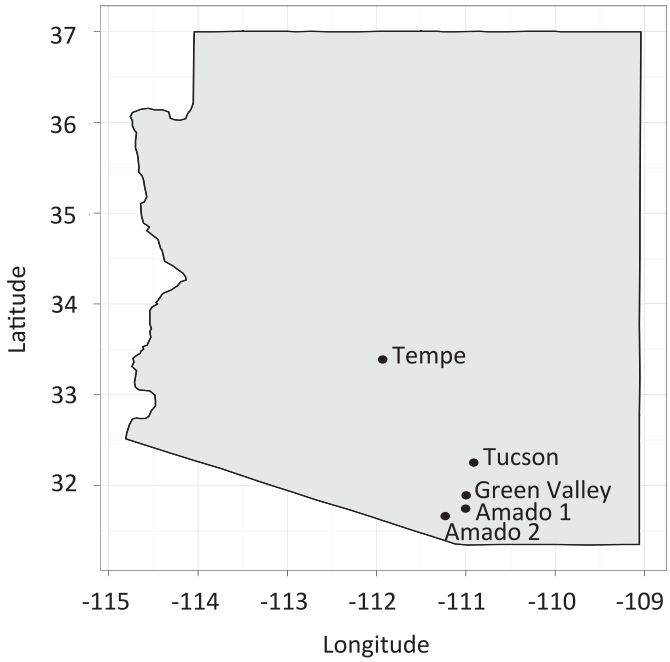

Fig. 1. Central Arizona (Phoenix-Tempe; 33.42°N, 111.93°W) and southern Arizona (Tucson 32.46°N, 110.94°W; Green Valley 31.87°N, 110.96; Amado 31.66°N, 111.23°W and 31.75°N).

cases have been more difficult to verify because of the higher prevalence of avian pox virus in western house finch populations, which like MG can cause swelling around the eyes (10). Previous studies have estimated that avian pox virus infections are present in approximately 20% of western house finches *vs.* less than 5% of eastern house finches (7). However, MG infection of house finches west of the Rocky Mountains was confirmed through PCR-based testing of house finches caught in Montana in 2002. Subsequently, DNA and culture-based evidence confirmed the presence of MG in the Pacific Northwest and California in the early to mid-2000s (10,13,18,24). Yet, reports of MG in the arid southwest regions of the United States east of California have not been confirmed by direct sampling. Here, we present results of house finch sampling in two areas of Arizona, southern and central portions of the state, presumably the leading edge of MG spread.

MATERIALS AND METHODS

House finch sampling. Following reports on house finch mortality associated with conjunctivitis in late 2009 to early 2010 in Arizona (4), in August 2011 we sampled Arizona house finches for evidence of MG infection. Our goal was to sample the Arizona Sun Corridor mega-region (Phoenix-Tempe and Tucson metropolitan areas) to determine whether MG was present and to determine the geographic extent of the disease. Birds with conjunctivitis-like signs were reported in Tucson and Green Valley (4), whereas regular monitoring since 2004 of house finches in the Phoenix-Tempe area produced no reports of MG-like conjunctivitis (McGraw, pers. obs.). We resampled these populations in July 2014 to examine temporal changes in MG prevalence and distribution.

In August 2011, we trapped house finches at bird feeding stations in southern Arizona (Tucson 32.46°N, 110.94°W and Green Valley 31.87°N, 110.96°W) and central Arizona (Phoenix-Tempe 33.42°N, 111.93°W). We trapped 21, 69, and 35 house finches in Tucson, Green Valley, and Tempe, respectively (20). Tucson is approximately 180 km southeast of Tempe and 50 km north of Green Valley (Fig. 1). In July 2014, we sampled 50 finches from Tempe, 66 from Tucson, 32 from Green Valley, and 61 from two additional southern Arizona locations near Amado (ca. 20 km south of Green Valley; 31.66°N, 111.23°W and 31.75°N, 111.00°W; Fig. 1).

Table 1. Results from 2011 and 2014 house finch sampling in southern and central Arizona.

Year	Sampling location	Conjunctivitis-like signs	No. PCR positive	No. antibody positive
2011	Southern Arizona			
	Tucson	2/21	0/21	—
	Green Valley	10/69	8/69	—
	Central Arizona			
	Tempe (Phoenix)	0/35	0/35	—
2014	Southern Arizona			
	Tucson	0/32	0/32	—
	Green Valley	0/66	0/66	5/50
	Amado 1	0/25	0/25	—
	Amado 2	0/36	0/36	—
	Central Arizona			
	Tempe (Phoenix)	3/50	0/50	—

Upon capture, a choanal swab was collected from each bird to test for the presence of MG (Roberts et al. 2001). In addition, birds were assessed for conjunctivitis-like signs (i.e., eye swelling) associated with MG infection (14). Because of a lack of observed conjunctivitis in 2014, we collected blood from a subset of 50 birds in Tucson and tested the serum for MG antibodies by serum plate agglutination using a commercially available MG Plate Antigen (material no. 10100760, Charles River Laboratories, North Franklin, Connecticut, USA) (31).

MG presence. Choanal swabs were tested for the presence of MG via end-point PCR and amplicon detection using agarose gel electrophoresis and staining with ethidium bromide (31). In brief, swabs were placed in 100 µl of sterile nuclease-free water. Swabs were then placed at 100 C for 10 min, at −20 C for 10 min, and finally centrifuged at 13,000 rpm for 5 min. We tested the supernatant of each sample in duplicate for MG presence using the forward primer 5′-GCTTCCTTGCGGTTAG-CAAC-3′ and reverse primer 5′-GAGCTAATCTGTAAAGTTGGTC-3′. PCR parameters were as follows: 94 C for 5 min; 35 cycles of 94 C for 30 sec, 55 C for 30 sec, and 72 C for 30 sec; and a final 5-min extension at 72 C (31). In each assay, MG DNA extracted from pure culture served as a positive control. Isolation in pure culture, although preferable, was not possible because of study constraints.

RESULTS

In 2011, 10 of 69 house finches from Green Valley and 2 of 21 house finches from Tucson had conjunctivitis-like signs. Of these 12 birds, only seven individuals, all from Green Valley, were confirmed positive for MG via PCR of the choanal swabs. In addition, one asymptomatic bird from Green Valley tested positive for MG via PCR, yielding 8 (11.6%) of 69 infected birds captured in Green Valley (Table 1). In contrast, in 2014 no birds sampled from Tucson, Green Valley, or Amado exhibited conjunctivitis or were PCR positive for MG based on choanal swabs. Of the 50 individuals from Tucson whose serum was tested for MG antibodies by rapid plate agglutination, only five (10%) were found positive. No individuals were found to have conjunctivitis in Tempe in 2011, whereas three birds exhibited suspect conjunctivitis-like signs in 2014. However, all birds sampled in Tempe tested negative for MG

by PCR (Table 1). Thus, no cases of MG infection have been confirmed in the Phoenix-Tempe metropolitan area to date.

DISCUSSION

Our study found evidence for MG in southern Arizona in 2011. Although we did not sample these populations before 2011 or at neighboring sites (i.e., along a disease-transmission path), it is possible that MG arrived at Green Valley via the leading edge of its spreading range from the east rather than from California in the west. By contrast, finches from central Arizona—specifically the Phoenix-Tempe region—appear to remain unexposed to MG as of 2014.

The absence of MG in Tempe, approximately 180 km north of where MG is known to be present (Green Valley), could be because of the lack of urban habitat between house finch populations in this arid region (i.e., along the Arizona Sun Corridor), hence reducing the opportunity for transmission. MG transmission requires direct exposure to MG-containing moisture droplets (9). Shared, anthropogenic resources such as bird feeders were thought to play a major role in MG's initial epizootic spread (1,11), in part by facilitating higher host abundance (22). However, despite high abundance of western house finches, in some populations there is no or only short-distance (<10 km) migration of house finches (although there is potential for contact between populations via juvenile dispersal) (4,5). For example, house finches from geographically adjacent urban habitats (University of Arizona, Tucson) and undisturbed natural Sonoran Desert habitats (Saguaro West National Park) display divergence in traits related to bill development and bite force (5). The divergence between these populations, separated by only 25 km, has been attributed to a lack of gene flow combined with selection resulting from differences in available food resources because of human provisioning (5). Based on 12 microsatellite loci, these same populations exhibit numerically small ($F_{ST} = 0.003$) but highly statistically significant genetic differentiation at levels typical of house finch populations separated by much greater distances (>800 km) in other parts of North America (5). Therefore, if we also assume lack of gene flow between Green Valley and Tempe birds, MG may not be able to spread to central Arizona house finches from the south.

Alternatively, the failure of MG to spread to Tempe may be a consequence of the virulence of the MG isolates currently in circulation. Indeed, models indicate that MG isolates from the disease front are likely to be of lower virulence, potentially reducing transmissibility, than those from regions where MG has become endemic (28). In agreement, a 2006 California MG isolate exhibited significantly lower virulence and transmissibility in house finches from eastern populations (34), which presumably have evolved MG resistance mechanisms (6), than a North Carolina 2006 isolate (34). However, by 2010 MG isolates circulating in California had demonstrably increased in virulence to a level comparable to an early epizootic Virginia 1994 MG isolate (19). Given that in August 2011, 2 yr after MG was initially reported in Arizona, approximately 12% of house finches in Green Valley were found to be infected with MG, it would suggest that reduced MG virulence was not a major factor inhibiting MG's spread to central Arizona. This could be confirmed in future studies, for example, by obtaining isolates in culture and their subsequent use in experimental infection studies or whole genome comparisons.

Contrary to what would be expected given the observed incidence of MG in Green Valley in 2011, we trapped no birds with active MG infections at the same site in July 2014. Whereas May–July MG occurrence is low in eastern and Montana house finch populations (3,10), in Arizona MG is thought to be most prevalent during spring and summer (4). Thus, possible explanations for change in prevalence of MG include 1) annual fluctuations in MG infections, 2) MG is no longer present in house finches from Green Valley, 3) limited sampling, or 4) sensitivity of detection methods (i.e., use of choanal swabs only rather than in combination with conjunctival swabs or culture). Just as low pathogen virulence may inhibit disease transmission, too high of virulence may also inhibit transmission (29). Initial reports on MG in Arizona from 2009 to 2010 indicated MG infections induced up to 70% local adult house finch mortality (4). If infected birds died without transmitting the disease, MG prevalence may have rapidly diminished (29). To clarify the underlying reason for the apparent absence of MG in Arizona in July 2014, continued and more thorough monitoring of southern Arizona house finch populations is necessary.

Although we have confirmed the presence of MG in southern Arizona, it is important to note that additional birds in all sampling locations were found to have some degree of eye swelling that was not attributable to MG (i.e., they tested negative for MG DNA). The difference in clinical presentation in these birds *vs.* known MG-infected birds suggests another pathogen, such as avian pox virus, may be responsible for the observed clinical signs. Avian pox viruses can cause swollen lesions on the eyes (in addition to other areas of the body) and thus be mistaken for MG-associated conjunctivitis (10,30,33). However, unlike MG, pox lesions typically have a dry or crusty appearance rather than a watery discharge, and they are occasionally bloody, as transmission of avian pox virus occurs predominantly through insect vectors and skin abrasions. In addition, pox lesions that occur on or around the eye are typically localized to a particular ocular region, whereas during an MG infection the entire conjunctiva tends to become inflamed (9,33). From 2000 to 2002, before MG was detected in the Pacific Northwest and long before MG reached California or the southwestern United States, house finches with conjunctivitis-like signs were reported in all of these regions through the House Finch Disease Survey and Project Feederwatch (10). After MG's arrival in Montana, reports of conjunctivitis increased drastically, suggesting that the previously reported instances of conjunctivitis-like signs may have been misidentified as MG (10). Although our sampling methods were insufficient for a definitive diagnosis, incorporation of protocols for testing for avian pox viruses (16,26) or other possible pathogens could help estimate the proportion of reported conjunctivitis cases that can be attributed to MG *vs.* avian pox and thus help clarify the pattern of MG occurrence across western North America.

In conclusion, our findings confirm the presence of MG in southern Arizona and provide further detail regarding the detection of MG in the southwestern United States. Furthermore, our data support the persistence of a MG-unexposed house finch population in central Arizona. These populations can continue to be an invaluable resource for studying host-pathogen coevolution in this system.

REFERENCES

1. Adelman, J. S., A. W. Carter, W. A. Hopkins, and D. M. Hawley. Deposition of pathogenic *Mycoplasma gallisepticum* onto bird feeders: host

pathology is more important than temperature-driven increases in food intake. Biol. Lett. 9:20130594. 2013.

2. Adelman, J. S., L. Kirkpatrick, J. L. Grodio, and D. M. Hawley. House finch populations differ in early inflammatory signaling and pathogen tolerance at the peak of *Mycoplasma gallisepticum* infection. Am. Nat. 181:674–689. 2013.

3. Altizer, S., W. M. Hochachka, and A. A. Dhondt. Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American house finches. J. Anim. Ecol. 73:309–322. 2004.

4. Badyaev, A. V., V. Belloni, and G. E. Hill. House finch (*Haemorhous mexicanus*). In: A. Poole, ed. The birds of North America online. Cornell Lab of Ornithology, Ithaca, NY. 2012.

5. Badyaev, A. V., R. L. Young, K. P. Oh, and C. Addison. Evolution on a local scale: developmental, functional, and genetic bases of divergence in bill form and associated changes in song structure between adjacent habitats. Evolution 62:1951–1964. 2008.

6. Bonneaud, C., S. Balenger, A. F. Russell, J. Zhang, G. E. Hill, and S. V. Edwards. Rapid evolution of disease resistance is accompanied by functional changes in gene expression in a wild bird. Proc. Natl. Acad. Sci. U. S. A. 108:7866–7871. 2011.

7. Davis, A. K., W. R. Hood, and G. E. Hill. Prevalence of blood parasites in eastern versus western house finches: are eastern birds resistant to infection? EcoHealth 10:290–297. 2013.

8. Delaney, N. F., S. Balenger, C. Bonneaud, C. J. Marx, G. E. Hill, N. Ferguson-Noel, P. Tsai, A. Rodrigo, and S. V. Edwards. Ultrafast evolution and loss of CRISPRs following a host shift in a novel wildlife pathogen, *Mycoplasma gallisepticum*. PLoS Genet. 8:e1002511. 2012.

9. Dhondt, A. A., S. Altizer, E. G. Cooch, A. K. Davis, A. Dobson, M. J. Driscoll, B. K. Hartup, D. M. Hawley, W. M. Hochachka, P. R. Hosseini, C. S. Jennelle, G. V. Kollias, D. H. Ley, E. C. Swarthout, and K. V. Sydenstricker. Dynamics of a novel pathogen in an avian host: mycoplasmal conjunctivitis in house finches. Acta Trop. 94:77–93. 2005.

10. Dhondt, A. A., A. V. Badyaev, A. P. Dobson, D. M. Hawley, M. J. L. Driscoll, W. M. Hochachka, and D. H. Ley. Dynamics of mycoplasmal conjunctivitis in the native and introduced range of the host. EcoHealth 3:95–102. 2006.

11. Dhondt, A. A., K. V. Dhondt, D. M. Hawley, and C. S. Jennelle. Experimental evidence for transmission of *Mycoplasma gallisepticum* in house finches by fomites. Avian Pathol. 36:205–208. 2007.

12. Dhondt, A. A., D. L. Tessaglia, and R. L. Slothower. Epidemic mycoplasmal conjunctivitis in house finches from eastern North America. J. Wildl. Dis. 34:265–280. 1998.

13. Duckworth, R. A., A. V. Badyaev, K. L. Farmer, G. E. Hill, and S. R. Roberts. First case of *Mycoplasma gallisepticum* infection in the western range of the house finch (*Carpodacus mexicanus*). Auk 120:528–530. 2003.

14. Farmer, K. L., G. E. Hill, and S. R. Roberts. Susceptibility of a naive population of house finches to *Mycoplasma gallisepticum*. J. Wildl. Dis. 38:282–286. 2002.

15. Fischer, J. R., D. E. Stallknecht, M. P. Luttrell, A. A. Dhondt, and K. A. Converse. Mycoplasmal conjunctivitis in wild songbirds: the spread of a new contagious disease in a mobile host population. Emerg. Infect. Dis. 3:69–72. 1997.

16. Gyuranecz, M., J. T. Foster, A. Dan, H. S. Ip, K. F. Egstad, P. G. Parker, J. M. Higashiguchi, M. A. Skinner, U. Hofle, Z. Kreizinger, G. M. Dorrestein, S. Solt, E. Sos, Y. J. Kim, M. Uhart, A. Pereda, G. Gonzalez-Hein, H. Hidalgo, J. M. Blanco, and K. Erdelyi. Worldwide phylogenetic relationship of avian poxviruses. J. Virol. 87:4938–4951. 2013.

17. Hartup, B. K., H. O. Mohammed, G. V. Kollias, and A. A. Dhondt. Risk factors associated with mycoplasmal conjunctivitis in house finches. J. Wildl. Dis. 34:281–288. 1998.

18. Hawley, D. M., K. V. Dhondt, A. P. Dobson, J. L. Grodio, W. M. Hochachka, D. H. Ley, E. E. Osnas, K. A. Schat, and A. A. Dhondt. Common garden experiment reveals pathogen isolate but no host genetic diversity effect on the dynamics of an emerging wildlife disease. J. Evol. Biol. 23:1680–1688. 2010.

19. Hawley, D. M., E. E. Osnas, A. P. Dobson, W. M. Hochachka, D. H. Ley, and A. A. Dhondt. Parallel patterns of increased virulence in a recently emerged wildlife pathogen. PLoS Biol. 11:e1001570. 2013.

20. Hill, G. E. A red bird in a brown bag: the function and evolution of colorful plumage in the house finch. Oxford University Press, Oxford, New York. 2002.

21. Hochachka, W. M., and A. A. Dhondt. Density-dependent decline of host abundance resulting from a new infectious disease. Proc. Natl. Acad. Sci. U. S. A. 97:5303–5306. 2000.

22. Hosseini, P. R., A. A. Dhondt, and A. P. Dobson. Spatial spread of an emerging infectious disease: conjunctivitis in house finches. Ecology 87:3037–3046. 2006.

23. Ley, D. H., J. E. Berkhoff, and J. M. McLaren. *Mycoplasma gallisepticum* isolated from house finches (*Carpodacus mexicanus*) with conjunctivitis. Avian Dis. 40:480–483. 1996.

24. Ley, D. H., D. S. Sheaffer, and A. A. Dhondt. Further western spread of *Mycoplasma gallisepticum* infection of house finches. J. Wildl. Dis. 42:429–431. 2006.

25. Luttrell, M. P., J. R. Fischer, D. E. Stallknecht, and S. H. Kleven. Field investigation of *Mycoplasma gallisepticum* infections in house finches (*Carpodacus mexicanus*) from Maryland and Georgia. Avian Dis. 40:335–341. 1996.

26. Manarolla, G., G. Pisoni, G. Sironi, and T. Rampin. Molecular biological characterization of avian poxvirus strains isolated from different avian species. Vet. Microbiol. 140:1–8. 2010.

27. Nolan, P. M., G. E. Hill, and A. M. Stoehr. Sex, size, and plumage redness predict house finch survival in an epidemic. Proc. R. Soc. Lond. B Biol. Sci. 265:961–965. 1998.

28. Osnas, E. E., P. J. Hurtado, and A. P. Dobson. Evolution of pathogen virulence across space during an epidemic. Am. Nat. 185:332–342. 2015.

29. Park, M., C. Loverdo, S. J. Schreiber, and J. O. Lloyd-Smith. Multiple scales of selection influence the evolutionary emergence of novel pathogens. Philos. Trans. R. Soc. Lond. B Biol. Sci. 368:20120333. 2013.

30. Parker, P. G., E. L. Buckles, H. Farrington, K. Petren, N. K. Whiteman, R. E. Ricklefs, J. L. Bollmer, and G. Jimenez-Uzcategui. 110 Years of *Avipoxvirus* in the Galapagos Islands. PloS One 6:e15989. 2011.

31. Roberts, S. R., P. M. Nolan, and G. E. Hill. Characterization of *Mycoplasma gallisepticum* infection in captive house finches (*Carpodacus mexicanus*) in 1998. Avian Dis. 45:70–75. 2001.

32. Roberts, S. R., P. M. Nolan, L. H. Lauerman, L. Q. Li, and G. E. Hill. Characterization of the mycoplasmal conjunctivitis epizootic in a house finch population in the southeastern USA. J. Wildl. Dis. 37:82–88. 2001.

33. Weli, S. C., and M. Tryland. Avipoxviruses: infection biology and their use as vaccine vectors. Virol. J. 8:49. 2011.

34. Williams, P. D., A. P. Dobson, K. V. Dhondt, D. M. Hawley, and A. A. Dhondt. Evidence of trade-offs shaping virulence evolution in an emerging wildlife pathogen. J. Evol. Biol. 27:1271–1278. 2014.

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