

Original Article

PATHOGENS AND PARASITES OF FERAL AND MANAGED HONEY BEE, *APIS MELLIFERA*, COLONIES FROM UTAH, USA

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Received: 26 September 2025; accepted: 05 February 2026

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Abstract

Honey bee colonies in the United States continue to experience health challenges driven by parasites, pathogens, and environmental stressors. To establish a statewide baseline for honey bee health in Utah, we conducted a multi pathogen and parasite survey of 429 feral and managed *Apis mellifera* colonies collected from seventeen counties across three major ecological regions between 2008 and 2021. Colonies were screened for *Varroa destructor*, *Nosema apis*, *N. ceranae*, *Lotmaria passim*, *Crithidia mellificae*, and the bacterial symbionts *Spiroplasma apis* and *S. melliferum*. Overall pathogen and parasite occurrence in Utah was low: *N. ceranae* and *L. passim* were each detected in 7.3% of colonies, while *V. destructor* was found in 9.0% of colonies screened. No samples were positive for *N. apis*, *Crithidia* spp., or either *Spiroplasma* species. Pathogen prevalence did not differ significantly between feral and non-migratory managed colonies, but strikingly low *Varroa* levels were observed in A-lineage feral colonies, with only one of forty-four colonies (2.3%) testing positive. Pathogens and parasites were detected across all ecological regions, although prevalence varied geographically and among mitochondrial DNA lineages. These results provide the first comprehensive assessment of pathogens and parasites in Utah honey bees, establish a critical baseline for future monitoring, and highlight the need for targeted studies investigating the potential mite resistance or reduced exposure of Utah's A-lineage feral populations.

Keywords: *Apis mellifera*, molecular diagnostics, parasite, pathogen, Utah

INTRODUCTION

Managed and feral honey bee colonies in the United States have experienced substantial declines in health over the past several decades due to a combination of biotic and abiotic stressors (vanEngelsdorp et al., 2009). Recent national surveys underscore the severity of these losses; for example, between 2019 and 2020 U.S. beekeepers reported a 43% reduction in colony numbers (Bruckner et al., 2023), and early reports from 2025 indicate that more than 60% of commercial colonies were lost within a single season, representing approximately 1.7 million colonies and an economic impact approaching USD 600 million (Lamas et al., in press). These recurring losses highlight the need for detailed, region-specific assessments of parasites and pathogens that threaten honey bee health. Several microbial and arthropod pathogens contribute directly to colony decline. The ectoparasitic mite *Varroa destructor* Anderson

and Trueman weakens honey bees by feeding on fat body tissue and by transmitting viruses such as deformed wing virus and acute bee paralysis virus (Ramsey et al., 2019; Posada-Florez et al., 2020). Microsporidia of the genus *Nosema* Nägeli, particularly *N. ceranae* (Fries et al.), are associated with digestive dysfunction, reduced productivity, and increased winter mortality (Botías et al., 2013; Rangel et al., 2016). Two trypanosomatid species, *Crithidia mellificae* Langridge and McGhee and *Lotmaria passim* Schwarz, commonly inhabit the honey bee hindgut and may contribute to reduced colony performance, while two *Spiroplasma* species, *S. apis* Mouches and *S. melliferum* Clark, invade the gut lumen and hemolymph and have been linked to colony stress events (Mouches et al., 1983; Clark et al., 1985; Tripodi et al., 2018). Despite the recognized importance of these pathogens, state-level surveys remain uncommon in the United States, and most existing studies focus on migratory commercial operations

rather than regionally stable, non-migratory colonies. Feral colonies in particular represent an understudied component of honey bee populations, yet they may provide insight into natural pathogen resistance, environmental influences on disease dynamics, and the long-term health of unmanaged populations. Several state-specific studies, including those conducted in Oklahoma, Arkansas, and Texas, demonstrate the value of localized surveys for understanding pathogen prevalence, lineage associations, and the potential influence of regional beekeeping practices (Herrera et al., 2017; Cleary et al., 2022; Szalanski & Cleary, 2025). Until now, however, no comprehensive assessment of parasites and pathogens has been conducted for honey bees in Utah.

The state of Utah is 219,887 km² in size and lies in the Mountain West subregion of the Western United States. It is divided into twenty-nine counties and into three distinct ecological regions (Rudis, 1999). Colonies of honey bees were first brought to Utah in covered wagons in 1848, and by 1872 there were approximately 2,000 honey bee colonies in Utah (Nye, 1976). Currently representatives of all major mitochondrial lineages (A, C, M, and O) occur in Utah (Cleary et al., 2018). Africanized honey bees were first officially discovered in southern Utah in 2008 (Hodgson et al., 2010), which constitutes the northern limits of their range in the US. Based on niche modeling evidence, most of southern to central Utah is suitable habitat for Africanized honey bees (Stohlgren et al., 2014).

The goal of this study was to conduct the first statewide evaluation of parasites and pathogens in Utah honey bees. Specifically, we aimed to: (1) determine the presence and distribution of *Nosema ceranae*, *N. apis*, *Lotmaria passim*, *Crithidia mellificae*, *Spiroplasma apis*, *S. melliferum*, and *Varroa destructor* in both feral and managed colonies; (2) examine pathogen occurrence across mitochondrial lineages; and (3) assess variation among Utah's distinct ecological regions. By integrating new molecular diagnostics with previously published lineage and pathogen data, this study establishes a comprehensive baseline for honey bee health in Utah and

provides a foundation for future monitoring and management strategies.

MATERIAL AND METHODS

Adult worker honey bees were collected from feral and managed colonies across seventeen Utah counties between 2008 and 2021 (Tab. 1). Following Schiff et al. (1994), feral colonies were defined as established colonies nesting in such unmanaged sites as tree cavities, caves, and structures. Managed colonies were obtained from non-migratory beekeepers within the state. All samples were preserved in 70-100% ethanol and shipped to the Insect Genetics Laboratory at the University of Arkansas (Fayetteville, AR, USA) for analysis. Because sample sizes varied across collections, *V. destructor* screening was conducted on 200 of the 429 available honey bee samples, following Cleary & Szalanski (2022). *Varroa* detection utilized a mite-wash protocol adapted from Oliver (2013), which involved agitating adult bees in alcohol and inspecting wash residues for the presence of mites.

For each honey bee colony, pooled DNA extractions were performed using between six and ten adult workers. Bees were removed from ethanol and air-dried for approximately three hours at 22°C to eliminate residual preservative. DNA was extracted with the use of a salting-out protocol with in-house reagents (Sambrook & Russell, 2001) following procedures outlined by Szalanski et al. (2016). Extracted DNA was stored at -20°C until use. Successful extraction was verified through PCR amplification of the mitochondrial COI–COII region with the use of primers E2 and H2 (Garnery et al., 1993; Cleary et al., 2018). Lineage identification incorporated both previously published and newly generated data. For 174 colonies previously analyzed for mitochondrial variation (Cleary et al., 2018), lineage assignments were retained based on COI–COII sequencing. Of the remaining 255 samples, thirty-one colonies were sequenced following the same methods, while 224 colonies were assigned to mitochondrial lineage with the use of PCR–RFLP analysis of the cytochrome-b gene (Ferreira et al., 2009), with fragment profiles

Table 1.

Utah counties sampled for *Nosema ceranae*, *Lotmaria passim*, and *Varroa destructor*

County	Samples	Lineage(s)	Source (n)	<i>N. ceranae</i>	<i>L. passim</i>	<i>V. destructor</i>
Cache	36	C(36)	M(36)	12(-), 2(+)	14(-), 0(+)	21(-), 0(+)
Carbon	12	C(12)	M(2), F(10)	5(-), 0(+)	11(-), 0(+)	10(-), 1(+)
Davis	1	C(1)	M(1)	-	-	1(-), 0(+)
Emery	50	C(46), A(4)	M(6), F(44)	39(-), 0(+)	30(-), 0(+)	41(-), 7(+)
Garfield	1	C(1)	F(1)	1(-), 0(+)	-	1(-), 0(+)
Grand	24	C(24)	M(3), F(21)	14(-), 7(+)	19(-), 2(+)	7(-), 4(+)
Iron	53	C(18), A(31), M(1), O(3)	M(9), F(44)	48(-), 5(+)	51(-), 2(+)	-
Juab	1	C(1)	M(1)	-	-	1(-), 0(+)
Kane	10	C(7), A(2), M(1)	M(6), F(4)	8(-), 0(+)	6(-), 0(+)	2(-), 0(+)
Millard	6	C(6)	M(5), F(1)	5(-), 0(+)	6(-), 0(+)	-
Salt Lake	10	C(10)	M(8), F(2)	7(-), 0(+)	3(-), 0(+)	10(-), 0(+)
San Juan	92	C(63), A(25), M(2), O(2)	F(92)	55(-), 3(+)	54(-), 2(+)	73(-), 6(+)
Sevier	1	C(1)	F(1)	1(-), 0(+)	1(-), 0(+)	-
Utah	5	C(5)	M(2), F(3)	3(-), 0(+)	1(-), 0(+)	2(-), 0(+)
Washington	124	C(60), A(57), M(2), O(5)	M(8), F(116)	120(-), 4(+)	78(-), 16(+)	10(-), 0(+)
Wasatch	1	C(1)	F(1)	1(-), 0(+)	1(-), 0(+)	-
Weber	2	C(2)	M(1), F(1)	1(-), 1(+)	2(-), 0(+)	2(-), 0(+)
Total	429	C(280), A(131), O(11), M(5)	M(88), F(341)	312(-), 22(+)	278(-), 22(+)	182(-), 18(+)

interpreted according to Taylor & Szalanski (1999). This approach enabled consistent lineage classification across all samples. A total of 334 samples were screened for *Nosema* spp. For 119 of these samples, existing diagnostic data from Szalanski et al. (2014) were incorporated. The remaining 215 samples were analyzed with the use of PCR amplification of the small subunit rRNA gene with primers *Nosema*SSU-1F and *Nosema*SSU-1R (Szalanski et al., 2014). Species-level identification (*N. apis* vs. *N. ceranae*) was conducted with the use of PCR-RFLP following Szalanski et al. (2014) and Szalanski & Cleary (2025). Detection of *Spiroplasma apis* and *S. melliferum* employed a multiplex PCR assay with primers *S.apis* ITS-F, *S.apis* ITS-R, Ms-160F, and Ms-160R (Schmid-Hempel & Tognazzo, 2010; Schwarz et al., 2014). Diagnostic interpretation followed methods described in Cleary & Szalanski (2024). Trypanosome data for 258 samples were obtained from Williams et al. (2024). For the remaining forty-two samples, a multiplex PCR assay targeting the CBSSU rRNA gene (Schmid-Hempel & Tognazzo, 2010) and the *L. passim*-specific 18S rRNA marker (Szalanski et al., 2016) was used to detect *Crithidia mellificae* and

Lotmaria passim.

All statistical analyses were performed in R version 4.0.5 (R Core Team, 2021). The relative proportions of colonies positive for *Nosema ceranae*, *Lotmaria passim*, *Spiroplasma* spp., and *Varroa destructor* were compared between feral and managed colonies and among mitochondrial lineages with the use of two-sample Z-tests with continuity correction. Similar comparisons were made between Utah pathogen prevalence and estimates reported for other U.S. states in previously published statewide surveys. Analyses were conducted with the use of sample sizes indicated in Tabs. 1-3.

RESULTS

A total of 429 honey bee colonies were analyzed from seventeen Utah counties, comprising 341 feral colonies (79.5%) and eighty-eight managed colonies (20.5%) (Tab. 1). Samples represented all three major ecological regions of the state (Rudis, 1999): the Colorado Plateau Semi-Desert (CP; n=226), Intermountain Semi-Desert and Desert (IM; n=152), and Great Plains-Palouse Dry Steppe (GP; n=51) (Tab. 2). Mitochondrial lineage

Table 2.

Occurrence of *N. ceranae*, *L. passim*, and *V. destructor* from honey bees collected from the Great Plains-Palouse Dry Steppe (GP), Intermountain Semi-Desert and Desert (IM), and Colorado Plateau Semi Desert (CP) ecoregions of Utah

Ecoregion	Counties	Samples	Lineage(s)	Source	<i>N. ceranae</i>	<i>L. passim</i>	<i>V. destructor</i>
GP	4	51	C(51)	F(12), M(39)	19(-), 3(+)	29(-), 0(+)	34(-), 1(+)
IM	10	152	A(35), O(3), M(1), C(113)	F(117), M(35)	109(-), 12(+)	111(-), 4(+)	63(-), 11(+)
CP	3	226	A(96), M(4), O(8), C(118)	F(212), M(14)	184(-), 7(+)	138(-), 18(+)	85(-), 6(+)
Total	17	429	A(131), M(5), O(11), C(282)	F(341), M(88)	312(-), 22(+)	278(-), 22(+)	182(-), 18(+)

Table 3.

Occurrence of *N. ceranae*, *L. passim*, and *V. destructor* among feral and managed sampled Utah honey bee lineages

Lineage	Feral or Managed	Samples	<i>N. ceranae</i>	<i>L. passim</i>	<i>V. destructor</i>
A	Feral	122	102(-) 8(+)	98(-) 11(+)	43(-) 1(+)
A	Managed	9	9(-)	8(-) 1(+)	1(-)
C	Feral	205	145(-) 10(+)	114(-) 8(+)	93(-) 9(+)
C	Managed	77	42(-) 2(+)	43(-) 1(+)	43(-) 6(+)
M	Feral	4	4(-)	4(-)	1(-) 1(+)
M	Managed	1	1(-)	1(-)	ND
O	Feral	10	8(-) 2(+)	9(-) 1(+)	1(-) 1(+)
O	Managed	1	1(-)	1(-)	ND
Total		429	312(-) 22(+)	278(-) 22(+)	182(-) 18(+)
Feral		341	259(-) 20(+)	225(-) 20(+)	138(-) 11(+)
Managed		88	53(-) 2(+)	53(-) 2(+)	44(-) 8(+)

analysis showed that most colonies belonged to the C lineage (n=280, 65.4%), followed by the A lineage (n=131, 30.6%), with the O (n=11, 2.7%) and M (n=5, 1.3%) lineages occurring at lower frequencies (Tab. 3). No samples tested positive for the bacterial pathogens *Spiroplasma apis* or *S. melliferum*.

Nosema ceranae was detected in twenty-two of 334 colonies screened (7.3%), with positive detections distributed across seven counties (Tab. 1). Feral colonies accounted for most positives (20/22; 7.2%), while two managed colonies (3.6%) were infected; however, this difference was not statistically significant (Z=0.67, p=0.504). By mitochondrial lineage, eight positives occurred in A-lineage colonies, ten in C-lineage feral colonies, two in C-lineage managed colonies, and two in O-lineage feral colonies (Tab. 3). No colonies were positive for *N. apis*. Across ecological regions, *N. ceranae* prevalence was

highest in the GP region (13.6%), followed by the IM (9.9%) and CP (3.7%) regions (Tab. 2).

Lotmaria passim was detected in twenty-two colonies (7.3%) and occurred in four counties (Grand, Iron, San Juan, and Washington). Similar to the *Nosema* results, feral colonies accounted for most detections (20/22), with two positives occurring in managed colonies (Tab. 1). This difference was not significant (Z=0.88, p=0.380). Twelve positive samples derived from the A lineage, nine from the C lineage, and one from the O lineage (Tab. 3). No samples tested positive for *Crithidia spp.* In terms of ecological distribution, *L. passim* was detected in the IM (3.5%) and CP (11.5%) regions but not in the GP region (Tab. 2). *Varroa destructor* mites were found in eighteen of the 200 colonies screened (9.0%), with detections occurring in four counties (Tab. 1). Of these, eleven infestations were in feral colonies (7.4%) and eight in managed colonies (15.4%),

though the difference was not statistically significant ($Z=1.42$, $p=0.155$). Lineage analysis revealed that nine positives occurred in C-lineage feral colonies, six in C-lineage managed colonies, one in an A-lineage feral colony (haplotype A1e), one in an M-lineage feral colony, and one in an O-lineage feral colony (Tab. 3). *Varroa* was detected in all ecological regions: IM ($n=11$), CP ($n=6$), and GP ($n=1$) (Tab. 2). A notable pattern emerged among A-lineage colonies: only one of the forty-four feral A-lineage colonies screened (2.3%) tested positive for *V. destructor*. This low prevalence contrasts with higher infestation rates in other lineages and highlights substantial variation in *Varroa* occurrence across honey bee mitochondrial DNA lineages.

DISCUSSION

This survey provides the first statewide, multi-pathogen assessment of feral and managed honey bee colonies in Utah and reveals a pathogen landscape that differs from patterns reported elsewhere in the United States. Overall prevalence of *N. ceranae*, *L. passim*, and *V. destructor* was low, particularly among feral and A-lineage colonies. Because our diagnostics detect presence but not infection intensity, these values are conservative and may underestimate low-level infections. Comparisons with other states highlight Utah's distinct profile. In Oklahoma, Szalanski and Cleary (2025) reported higher pathogen pressure and clear differences between feral and managed colonies. In Texas, *N. ceranae* and *V. destructor* were detected in 71% and 76% of colonies, respectively (Herrera et al., 2017). In Arkansas, *N. ceranae* and *L. passim* were each found in 10.2% of colonies and *Varroa* in 49.4% (Cleary & Szalanski, 2022). Nationally, Abban et al. (2024) reported *Nosema* prevalence near 100% and *Varroa* prevalence of 85.4% in symptomatic colonies. In contrast, Utah's prevalence estimates for these pathogens are far lower, suggesting reduced pathogen pressure and emphasizing the importance of regional context.

Extensive feral honey bee populations are

documented in Utah, particularly in remote desert and high-elevation habitats (Stohlgren et al., 2014; Cleary et al., 2018; Tripodi et al., 2019). These colonies occur at low densities and often in rugged terrain, reducing contact with managed colonies. Utah also supports relatively few large commercial operations; most managed colonies are maintained in small, non-migratory apiaries (USDA-NASS 2021, Utah Department of Agriculture and Food 2024). Such landscape and management characteristics likely limit horizontal pathogen transmission. Studies show that colony spacing, apiary density, and isolation strongly influence *Varroa* and pathogen dynamics, with larger distances between colonies reducing mite movement and feral colonies with limited contact exhibiting lower pathogen prevalence (Seeley, 2007; Rangel et al., 2016; Nolan & Delaplane 2017). These findings support the interpretation that Utah's ecological and beekeeping context contributes to the low pathogen levels observed here.

The exceptionally low levels of *V. destructor* in A-lineage feral colonies are among the most notable findings. Only one A-lineage feral colony (2.3%) was *Varroa*-positive, consistent with reports that Africanized honey bees often exhibit lower mite infestation levels than European-derived lineages (Mondragón et al., 2005; Guzman-Novoa et al., 2012; Castilhos et al., 2023). Africanized bees display such traits as elevated grooming, efficient removal of infested brood, and shorter postcapping periods, which reduce mite survival and reproduction (Spivak & Gilliam, 1998; Arechavaleta-Velasco & Guzmán-Novoa, 2001; Vandame et al., 2002; Martin & Medina, 2004). Many A-lineage colonies in this survey occupy high-elevation or remote desert habitats, limiting contact with managed colonies that often act as *Varroa* reservoirs. Geographic isolation and reduced colony density are known to slow mite transmission (Chemurot et al., 2016; Nolan & Delaplane, 2017). Together, these ecological and behavioral factors provide plausible explanations for the very low mite prevalence in A-lineage feral colonies and highlight the need for targeted research on potential resistance mechanisms.

In regions with intensive migratory beekeeping,

managed colonies often experience high disease pressure due to increased colony mixing, exposure to pathogen hotspots, and transport-related stress (Pettis et al, 2013; Traynor et al., 2016; Steinhauer et al., 2018). Migratory colonies can accumulate higher loads of *Nosema*, viruses, and *Varroa* than stationary colonies (Dainat et al., 2012; Cavigli et al., 2016; Smart et al., 2016). In contrast, Utah's managed colonies are predominantly non-migratory, which likely reduces pathogen spillover and helps to explain why managed colonies in this study did not exhibit greater pathogen prevalence than feral colonies. These differences underscore the value of state-level surveys: regional variation in climate, beekeeping practices, and colony density can produce pathogen patterns that differ markedly from national trends.

Because this study spans thirteen years, it offers a long-term perspective on pathogen presence in Utah honey bees. Integrating previously published diagnostics with new molecular screening allows comparisons across years using consistent methods and reveals no evidence of rapid pathogen expansion during the sampling period. Pathogen pressure appears relatively stable, particularly in feral populations that dominate Utah's honey bee landscape.

Several limitations should be considered. Feral colonies comprised most samples, limiting power to detect differences between management types. Some counties and regions were represented by relatively few colonies, which may obscure finer-scale patterns. Finally, PCR and PCR-RFLP are less sensitive than qPCR, so prevalence estimates are conservative. Future statewide surveys would benefit from incorporating qPCR-based diagnostics to quantify pathogen loads and to improve detection of low-intensity infections. Follow-up studies should include repeated sampling within the same colonies to assess temporal fluctuations and evaluate the stability of pathogen presence within lineages and regions. Behavioral or genomic investigations of Utah's feral A-lineage colonies are also warranted to determine whether these populations possess heritable mechanisms of *Varroa* resistance. Such efforts could contribute to broader national

initiatives aimed at identifying naturally resilient honey bee populations within the United States. Overall, this study enhances our understanding of honey bee health in a region historically underrepresented in bee pathology research. By documenting pathogen occurrence across lineages, management types, and ecological regions, the results establish an important baseline for continued monitoring. The exceptionally low *Varroa* levels observed in feral A-lineage colonies, in particular, highlight Utah as a critical region for future research into natural mechanisms of parasite tolerance or resistance.

ACKNOWLEDGEMENTS

We thank the beekeepers in the state of Utah along with Jerry Shue, Danielle Downey, Joey Caputo, and the Utah Department of Agriculture and Food for assisting with sample collection. This research was supported in part by the University of Arkansas, Arkansas Agricultural Experiment Station, and the open access publishing fund administered through the University of Arkansas Libraries.

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