Molecular epidemiology of a fatal sarcoptic mange epidemic in endangered San Joaquin kit foxes (*Vulpes macrotis mutica*)

Jaime L. Rudd [1, 2*], Deana L. Clifford [1,2], Brian L. Cypher [3], Joshua M. Hull [4], A. Jane Riner[1,2], And Janet E. Foley [1]

[1] Department of Medicine and Epidemiology, University of California, Davis, CA 95616, USA; [2] Wildlife Investigations Laboratory, California Department of Fish and Wildlife, Rancho Cordova, CA 95670, USA; [3] Endangered Species Recovery Program, California State University, Stanislaus, Turlock, CA 95382, USA; [4] United States Fish and Wildlife Service, Sacramento, CA 95825, USA

*Jaime.Rudd@wildlife.ca.gov

Abstract

Background: In 2013, sarcoptic mange, caused by *Sarcoptes scabiei* mites, precipitated a catastrophic decline of the formerly stable urban population of endangered San Joaquin kit foxes (*Vulpes macrotis mutica*) in Bakersfield, California, USA. In 2019, a smaller sarcoptic mange outbreak affected kit foxes 58 km southwest of Bakersfield in the town of Taft, California. To determine whether the Taft outbreak could have occurred as spillover from the Bakersfield outbreak and whether epidemic control efforts must involve not only kit foxes but also sympatric dogs (*Canis lupus familiaris*), coyotes (*Canis latrans*), and red foxes (*Vulpes vulpes*), we evaluated genotypes and gene flow among mites collected from each host species.

Methods: We used 10 *Sarcoptes* microsatellite markers (SARM) to perform molecular typing of 445 *S. scabiei* mites collected from skin scrapings from twenty-two infested kit foxes, two dogs, five coyotes, and five red foxes from Bakersfield, Taft, and other nearby cities.

Results: We identified 60 alleles across all SARM loci; kit fox- and red fox-derived mites were relatively monomorphic, while genetic variability was greatest in Bakersfield coyote- and dog-derived mites. AMOVA analysis documented distinct mite populations unique to hosts, with an overall F_{ST} of 0.467. The lowest F_{ST} (i.e. closest genetic relationship, $F_{ST} = 0.038$) was between Bakersfield and Taft kit fox-derived mites while the largest genetic difference was between Ventura coyote- and Taft kit fox-derived mites ($F_{ST} = 0.843$).

Conclusion: These results confirm the close relationship between the Taft and Bakersfield outbreaks. The *Sarcoptes* host specificity suggests that, although an inter-species spillover event likely initiated the kit fox mange outbreak, mite transmission is now primarily intraspecific. Therefore, any large-scale population level intervention should focus on treating kit foxes within the city.

Keywords: Bakersfield, California, management, host specificity, microsatellites, mites, S*arcoptes scabiei*, wildlife

Background

The first written reference to the skin disease now known as scabies dates to 1200 BCE [1]. From the time of the ancient Greeks and Romans through the middle ages, mange was known as the “itch” [2] and its cause was unknown until 1687, when the Italian physician Giovanni Cosimo Bonomo identified a mite as the causative agent, making scabies one of the

first diseases in human history with a known etiology [2]. In 1778, DeGeer formally named the itch mite *Acarus scabiei,* and this classification was revised in 1802 to a new genus, *Sarcoptes,* now with only a single species, *S. scabiei* [3, 1]. The World Health Organization (WHO) describes human scabies as a prevalent, contagious condition affecting more than 200 million people each year worldwide [4].

Sarcoptes scabiei is also the agent of sarcoptic mange in animals and affects over 100 mammalian species [3, 5]. Infested domestic animals may have been original sources of mites, which then crossed species boundaries and infested many wildlife hosts [6]. *Sarcoptes scabiei* now appears to be a single but highly variable mite species with many host-restricted genetic variants [7-13]. Although past assessment of host-variant relationships was limited to morphological examination [7], advances in DNA-based techniques now permit improved resolution of those relationships. Microsatellite studies in particular have revealed that, even when multiple species in a community are infested with sarcoptic mange, each may harbor its own host-restricted genetic variant [10]. Documentation of the host specificity of each variant can help determine the host that served as the original source of the epidemic, how many species are involved, and how best to intercede.

A continuing sarcoptic mange epidemic is causing a dramatic decline in a subpopulation of San Joaquin kit foxes (*Vulpes macrotis mutica*, kit fox hereafter). One-hundred years of widespread agricultural and urban development have extirpated these kit foxes throughout much of their historic range such that this subspecies of kit fox is now federally endangered [14], persisting in a small metapopulation of three main subpopulations and less than a dozen satellite subpopulations in the western and southern ends of the San Joaquin Valley in central California [14, 15]. Remarkably, the largest satellite subpopulation of kit foxes in the city of Bakersfield

was stable despite ongoing urbanization [16, 17] and was considered a possible source for reintroductions to hedge against catastrophic declines in natural lands [17, 18]. However, after the initial detection of mange in 2013 [19], disease spread rapidly throughout this urban subpopulation causing substantial mortality. More than 460 kit foxes have been infested as of October 2018, and all cases have been lethal if not treated [5, 19, Cypher personal communication]. In January 2019, sarcoptic mange was detected in a smaller sub-population of kit foxes living in the city of Taft, 58 km southwest of Bakersfield [Cypher unpublished data].

Kit foxes in these unique and valuable urban populations are relatively habituated to human presence and easily captured, and affected individuals can be treated in rehabilitation facilities [19]. However, more efficient population-level management, including prevention of the spread of mange, could have a greater impact than individual animal treatment. Without knowing whether kit foxes share mite variants with sympatric species such as coyotes (*Canis latrans*), dogs (*C. lupus familiaris*), or red foxes (*V. vulpes*), it is not known whether management strategies to address mange in kit foxes must incorporate the other hosts as well. Accordingly, the goal for this project was to use molecular epidemiology to determine if the Taft outbreak is part of the Bakersfield outbreak and to understand the extent to which sympatric species share *S. scabiei* mite variants with kit foxes. Such insight would be invaluable, as management strategies that target multiple canids would be considerably more difficult rather than a single species strategy primarily due to varying ecology, home range size, ease of capture for application of preventative treatment, and public acceptance of prevention programs (e.g. dogs vs. coyotes)[17, 20].

Methods

Collection of *Sarcoptes scabiei* mites

Sampling was opportunistic, consisting of animals that were found dead, euthanized for humane reasons, or euthanized because of threats to public safety or domestic animals. These included foxes found dead or euthanized due to mange provided by the Endangered Species Recovery Program (ESRP) and the California Department of Fish and Wildlife (CDFW), ill stray dogs from Kern County Animal Services and Bakersfield Animal Care Center, and coyotes found dead due to vehicular strike (at Fort Irwin), euthanized due to severe mange (Ventura), hit by vehicles, or euthanized for depredation by the United States Department of Agriculture Wildlife Services (USDA, Figure 1).

All carcasses were frozen at -20C and then transferred to the CDFW Wildlife Investigations Laboratory (WIL) for examination and sampling of mites. Carcasses were carefully examined for skin scaling, hair loss, pustules, papules, and hyperkeratosis along the tail, rear legs, dorsum, abdomen, elbows, forelegs, neck, and head. Skin at the periphery of suspected lesions was scraped with a sterile surgical blade; contents of the scraping were placed onto a clean microscope slide with 3 drops of sterile water and examined under 400X magnification for presence of mites. Mites were morphologically identified as *S. scabiei* [21] and 5cm x 5cm sections of mite-positive skin were excised and stored in sterile plastic bags. Because mites were rare on dog skin, positive skin scrapings from dogs were flushed from the glass slide into 2-ml microfuge tubes with 70% EtOH. Skin samples and microfuge tubes were stored at -20C.

Preparation of mite DNA and microsatellite analysis

Frozen skin and scraping contents were thawed and individual mites collected using microscopy. Each mite was pierced with a sterile 18-gauge needle under a dissecting microscope and digested overnight in lysis buffer and proteinase K (Qiagen, Valencia, CA, USA) at 56°C. The Micro DNA Extraction Kit (Qiagen) procedure was used for the preparation of mite DNA from each individual mite according to the manufacturer's recommendations. Final DNA from each mite was eluted in 60µl of buffer AE.

We used ten microsatellite markers (SARMS 33-38, 40, 41, 44, and 45; Table 1) to genotype mites with modifications to the published protocol [10]. Forward primers were labeled with HEX or 6-FAM dye (Integrated DNA Technologies, Coralville, IA, USA) and reconstituted into 100 µM working dilutions. Primer pairs were combined into paired multiplexes with 1.5 – 2.5 µM of each primer. We performed PCR using the Qiagen 2X Type-it Multiplex PCR Master Mix, 10X multiplex primer mix (2.5 µl), DNA-free water (7 µl), and 2-3 µl DNA for a total reaction of 25 µl. Thermocycling conditions were as published (Rasero et al. 2010). PCR products were transferred to 96-well plates (Biotix Inc, San Diego, CA, USA) for electrophoresis and measurement of length polymorphisms on an ABI 3730 analyzer (Thermo Fisher, CA, USA) at the Veterinary Genetics Laboratory (Davis, CA, USA) and output sequence alignments were automated using the program STRand [22]**.** Microsatellite allele scoring was performed with the R-package MSATALLELE [23]. Data were first organized in an EXCEL (Microsoft, Redmond, WA, USA) spreadsheet and converted into genepop format using the program CREATE [24]. Possible genotyping errors due to stuttering or large allele dropout were evaluated using MICROCHECKER [25]. Null alleles were estimated using ML-RELATE [26].

Genetic analysis for population differentiation

Initially, we genotyped 20 – 30 mites from each host individual, except where only one or two mites could be found (e.g. dogs). After genotyping 351 mites from 19 canids at 20-30 mites per individual, it was observed that mites from individual kit fox lacked genetic diversity, therefore subsequent analysis only incorporated 1-6 mites per kit fox host individual.

For mites from each host population, we estimated allelic richness (R), number of polymorphic loci, expected (H_e) and observed heterozygosity (H_o), Hardy-Weinberg equilibrium (HWE), linkage disequilibrium (LD), and partitioned components of variance using Analysis of Molecular Variance (AMOVA). To evaluate differentiation among the *S. scabiei* mite populations, we calculated the pairwise F_{ST} and visualized the differences using principal components analysis (PCA). Analyses were completed using the software GENALEX v. 6.2 [27] and R packages [28] PopGenReport [29], adegenet [30], and poppr [31]. p-values ≤ 0.05 were considered statistically significant. Origin of hosts was mapped using ArcGIS version 10.3.1 (ESRI, Redlands, CA, USA).

Results

We assessed population genetics of 445 *S. scabiei* mites from 41 host individuals in four host species, including six mites from two dogs from Bakersfield, 137 mites from seven coyotes (five from Bakersfield, one from Fort Irwin, and one from Ventura), 192 mites from 21 kit foxes from Bakersfield, 23 mites from five Taft kit foxes, and 87 mites from four red foxes from Bakersfield and one from Fresno (Table 2). Although occasional loci of particular mites did not amplify (Table S1), 60 alleles were detected across the 10 microsatellite loci of all four host species, ranging from two alleles detected at SARM-38 to 11 alleles at SARM-33. A total of 31

158 private alleles were detected, distributed among eight loci. All loci showed LD ($p = 0.001$) and
159 significant deviations from HWE ($p < 0.001$).

160

161 **Kit fox mites**

162 Among the Bakersfield kit fox-derived mites, despite relatively large numbers of mites, there
163 was low overall mean allelic richness ($R = 1.29$) and all except SARM-33, 40, 41, and 44 were
164 monomorphic (Table 3, Table S1). There were 15 alleles across the variable microsatellite loci
165 including a private allele at SARM-40 at a frequency of 0.003. Values of H_o (0.082) and H_e
166 (0.117) were relatively low. SARM-33, 40, and 41 were not in HWE, had possible null alleles at
167 frequencies of 0.135, 0.177, and 0.167 respectively, and were in LD ($p < 0.05$). Mites collected
168 from Taft kit foxes were similar to Bakersfield kit fox mites and also had low allelic richness (R
169 = 1.19). There were only two polymorphic loci, SARM-40 and 44 (Table 3), and a total of 12
170 alleles across all loci. No private alleles were detected and, similar to Bakersfield kit fox mites,
171 Ho (0.076) and He (0.085) were low. All alleles were in HWE and there was no LD.

172

173 **Red fox mites**

174 Red fox-derived mites from Bakersfield had multiple alleles only at the same three loci as kit
175 fox mites, whereas SARM-36 was also variable among the Fresno red fox mites (Table 3). A
176 single private allele was detected at SARM-41 at a frequency of 0.05 in mites from the Fresno
177 red fox and no private alleles were detected in mites from the Bakersfield red foxes. Mean R was
178 low for both Bakersfield and Fresno ($R = 1.28$) with 15 and 14 total alleles detected in these mite
179 populations, respectively. Values of H_e from both Bakersfield and Fresno resembled the kit fox

mites, as did H_o from Bakersfield, whereas H_o from Fresno was slightly lower (0.114). The only deviation from HWE was at SARM-33 in mites from Bakersfield ($p < 0.05$). A null allele was detected at SARM-33 at a frequency of 0.295. Both SARM-33 and SARM-40 were in LD ($p < 0.05$). Null alleles were not detected in mites from the Fresno red fox and there was no LD.

Domestic dog mites

Despite obtaining only three mites from each of the two dogs, only two loci (SARM-35 and 37) had fixed alleles with a total of 22 alleles detected (Table 3). Two private alleles were detected at SARM-34. Average allelic richness was 2.08 and dog mites had lower H_o (0.017) but higher H_e (0.368) than mites collected from foxes (Table 2). Only SARM-35 and SARM-37 were in HWE and null alleles were detected at SARM-33 and 34 at frequencies of 0.382 and 0.403, respectively. LD was detected between SARM-33 and 34, 36, 38, 40; SARM-34 and 36 and 40; SARM-36 and 38 and 40; SARM-38 and 40; and SARM-41 and 44 ($p < 0.05$).

Coyote mites

Similar to dogs, the majority of loci in mites from coyotes were variable, with only three fixed loci in mites from Ft. Irwin (SARM-36, 37, and 38) and none from Bakersfield (Table 3). However, all except SARM-34 and 44 were monomorphic in mites from Ventura. Total numbers of detectable alleles were much higher than for other mite populations, with 52 detectable alleles in Bakersfield coyotes. There were private alleles in Bakersfield coyotes at eight loci (SARM-33, 34, 35, 37, 40, 41, 44, and 45), a single private allele in Ft. Irwin at SARM-40, and a single private allele in Ventura at SARM-34. Mean R (2.78) was notably high in these Bakersfield

coyote mites whereas the lowest overall H_o (0.023) and H_e (0.032) occurred in the Ventura coyote mites. There was significant HWE departure for Ventura SARM-44 ($p = 0.01$) and for all loci in Bakersfield coyote mites ($p < 0.05$), but none in Ft. Irwin. Null alleles were detected at all loci except SARM-45 at frequencies ranging between 0.121 and 0.364 for Bakersfield coyote mites. Bakersfield coyote mites had LD at SARM-33 and 34, 36, 37, 38, 40, 41, 44, and 45; SARM-34 and 36, 38, and 44; SARM-35 and 45; SARM-36 and 38; SARM-37 and 38, 40, and 44; and SARM-40 and 44 ($p < 0.05$). LD was also detected in Ft. Irwin coyote mites between SARM-36 and 40 ($p < 0.05$).

Host Population Differentiation

AMOVA analysis showed significant differentiation among host species-derived mite populations ($F_{ST} = 0.468$, $p < 0.01$, Table 4, Figure 2). The most closely related populations were mites from Bakersfield and Taft kit foxes ($F_{ST} = 0.038$, $p < 0.01$), followed by mites from kit foxes and red foxes from Bakersfield ($F_{ST} = 0.05$, $p < 0.01$). The least related mites were from the Taft kit foxes and Ventura coyote ($F_{ST} = 0.843$, $p < 0.01$). Bakersfield kit fox and Bakersfield coyote mites were also genetically distinct (0.508, $p < 0.01$) as were Bakersfield coyote and dog mites (0.168, $p < 0.01$). These relationships were also clear on the scatterplot of the PCA of mites, on which Bakersfield and Taft mites clustered together, but were distinct from all other mites (Figure 2).

Discussion

223 *Sarcoptes scabiei* infests a wide array of hosts in many communities. Although multiple
224 hosts in a community may experience mange simultaneously, examination of mite genetics often
225 reveals various degrees of host preference and specificity – a phenomenon described as the
226 "host-taxon law" [6, 10, 11, 32-38]. Mange is fatal in kit foxes and may contribute to local
227 extinction of the endangered San Joaquin kit fox in Bakersfield [19]. Therefore, we investigated
228 the population genetic structure of *S. scabiei* mites among these host species in order to
229 understand the risk that coyotes, dogs, and red foxes pose for mite spillover into kit foxes, with
230 the ultimate goal of developing an intervention strategy. Our data clearly reveal host-specificity
231 and that kit foxes acquire mite infestations from other kit foxes, both in Bakersfield and in Taft.

232 Based on data from 10 microsatellite markers, the most important source of population
233 structure (46% on AMOVA) among the mites was the host and to a lesser degree geographical
234 source, although samples from geographical locations outside of Bakersfield were limited as this
235 was not the original intention of the study. Bakersfield mites from kit and red foxes, and mites
236 from Taft kit foxes, had relatively few alleles and low heterozygosity compared to dogs and
237 coyotes, comprising an obvious cluster differentiated from other host species regardless of
238 geographic area. However, red fox mites from Fresno were less closely related to the Bakersfield
239 or Taft fox mites, and regardless of location, mites from domestic dogs and coyotes were
240 intermixed, suggesting different epidemiological cycles of mange in different areas. The lack of
241 genetic variability and fixation of alleles among Bakersfield kit and red foxes is consistent with a
242 founder event in the Bakersfield fox population. Because the Bakersfield outbreak was reported
243 six years prior to the Taft outbreak, it is suspected that the source of mites in Taft was
244 Bakersfield. Further host genetic analysis of Bakersfield and Taft kit foxes could elucidate gene

flow between foxes in these locations, and additional camera or telemetry data could help clarify prospects for gene flow among the kit foxes and mites as well.

Thirteen of the 14 alleles detected in Bakersfield kit fox mites, all 12 alleles in Taft kit fox mites, and all 15 alleles in red fox mites from Bakersfield were also found in coyotes and domestic dogs. The Bakersfield kit fox-derived mites had one private allele but only at low frequencies which could be due to genotyping error. Presence of private alleles can indicate isolation of mite populations and host-associated genetic variants [11]. *S. scabiei* mites can be transmitted between red foxes and domestic dogs in Europe [10], supporting the likelihood that the original source of mange among Bakersfield kit foxes was coyotes or dogs. Further indication of an original spillover in Bakersfield and Taft with a founder event is the absence of polymorphic loci across 282 mites collected from 31 foxes (27 kit foxes and 4 red foxes) belonging to the same *Vulpes* genus. Bakersfield dog and coyote mites had high R, H_o and H_e consistent with a large ancestral population of mites as is described for other mites collected from the genus *Canis* [12, 40].

Multiple studies [11, 13, 39, 37, 38] have examined a relatively small number of mite individuals from a host individual, implicitly assuming that genetic variability of mites on a single host was negligible. We found little benefit to sampling more than 25-30 individual mites per host individual in our study, in part because common alleles tend to be more informative than rare alleles when evaluating genetic composition within a population [40], and also because there was so little genetic variability among mites from any kit fox individual. Moreover, in the case of dogs, we could not necessarily achieve our target sample size of 30 mites. Despite this limitation, the six mites we did collect from two dogs showed considerable heterozygosity and allelic richness.

Coyote mites from Bakersfield, while the most variable, also had significant deviations from HWE, were in LD, and may have had null alleles – findings similar to other *Sarcoptes* and mite studies [10, 39, 41]. Null alleles are found in most taxa and are especially prominent in insects and bi-valves, resulting in homozygous excess and deviations from HWE [42 – 44]. This suggests that we may be underestimating the total variability in the coyote mite population. Given the larger home range sizes of coyotes, it is also possible that the deviations from HWE could have occurred if locations of sampling of coyotes and their mites were different from the areas where they typically resided (e.g. Wahlund effect).

Conclusions

Microsatellites are commonly used to study recent evolutionary events and have often been used to study molecular genetics of mange, thus our use of microsatellites allows our results to be compared directly to findings in the literature. There are other molecular methods such as Radseq, which analyzes single nucleotide polymorphisms (SNPs), which may offer insight into finer scale phylogeographic patterns [45] and possible historic host-associated introductions. Further, use of SNPs could potentially avoid some of the biases and subjectivity that can impact allele scoring of microsatellites. Whether using microsatellites or whole next generation genotypical approaches, ongoing genetic surveillance will be necessary to detect rare cross-species transmission among southern San Joaquin Valley canids which could initiate mange emergence in exurban kit foxes, an event that could contribute to species-wide declines.

Scabies in humans is often associated with overcrowding such as in hospitals, nursing homes, prisons, and schools 46 – 50]. High population densities may underlie sarcoptic mange

epidemics in wildlife as well [51]. The Bakersfield kit fox population is uniquely dense relative to exurban populations and these kit foxes may share dens with skunks (*Mephitis mephitis*) and domestic cats (*Felis catus*) or occupy dens previously used by red foxes, coyotes, or feral dogs. Such a complex contact network with high density and high host biodiversity could exacerbate the mange epidemic; however, biodiversity per se appears unlikely to contribute given our finding of host-restricted mite variants. The high densities of Bakersfield kit foxes may contribute to the severe impact of mange on this population, which could suggest that less dense exurban populations may be at less risk. The existence of host-restricted mite variants could also explain why it is common to observe mange-infested animals living in a community with other, mange-free host species.

Large epidemics of sarcoptic mange with high case fatality rates can devastate free-ranging wildlife [52 – 56]. Spillover of mites from common and less clinically impacted species such as coyotes and domestic dogs poses an important conservation challenge for wildlife managers and can inhibit recovery efforts [57]. Therefore, documenting the true likelihood of acquiring infestation from conspecific and sympatric hosts is crucial for intervention to support the most at-risk species. Our data clearly document that kit fox mites circulate primarily among Bakersfield kit foxes with occasional transmission or spillover to and from red foxes. The recent Taft epidemic is closely related to the epidemic in Bakersfield kit foxes and probably derived from Bakersfield kit foxes directly. This suggests that efforts to control mange in the kit fox population should focus on kit foxes and that other species are not primarily involved in this epidemic.

Ethics Approval and consent to participate

Sample collection was opportunistic and performed with consent from study partners. Work was conducted under authorization of a 10(a)1(A) recovery permit TE825573-2 from the United States Fish and Wildlife Service, a Memorandum of Understanding from the California Department of Fish and Wildlife (CDFW), and University of California Davis Institutional Animal Care and Use protocol #18179.

Consent for publication

Not applicable.

Availability of data and materials

Data are available on request to the authors.

Competing interests

The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the CA Department of Fish and Wildlife or the U.S. Fish and Wildlife Service. The authors declare that they have no competing interests.

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Authors' contributions

JLR carried out sample collection, microsatellite typing, conducted the raw data analysis, and wrote the manuscript. DLC, BLC, and JMH, participated in study design and writing the manuscript. AJR processed the samples and prepared them for microsatellite analysis. JF participated in study design, data analysis, and writing the manuscript. All authors have read and approved the final manuscript.

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List of Tables

Supplementary information

Supplementary information accompanies this paper at [link here].

Additional file 1: Table S1. Distributions of allele frequencies in 10 microsatellite loci among *Sarcoptes scabiei* mite populations by host and host location (allele sizes are in base pairs). Private alleles are denoted with "†".

Figure 1. Map of mite-host sampling locations in California

Figure 2. Dispersion of dog-, coyote-, kit fox -, and red fox-associated mite populations according to principal components analysis (PCA)