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A spatial stochastic model simulating a scabies epidemic and coyote population dynamics

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Abstract

Scabies is an important disease that affects many species and can greatly reduce population sizes in some species, yet have little effect on populations of other species. Here, we develop an exploratory mechanistic model to examine scabies epidemiology in the context of host demographics. As a starting point, we use empirical estimates from a well-studied coyote population to generate realistic population structures and parameter values for host population demographics and scabies epidemiology. The purpose of this paper is to determine whether our empirical knowledge of coyote demography and scabies epidemiology is sufficient to reproduce the patterns observed, and to highlight those areas where discrepancies exist. Where we find discrepancies, we modify the model to ameliorate the fit to the empirical patterns, as a means of generating hypotheses. We suggest that exploratory excursions by territorial individuals are crucial for maintaining population stability under epidemic conditions. Further, we believe that host evolution probably occurred within the decade of the epidemic. We identify other areas that require further attention, both empirically and theoretically.

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1. Introduction

Sarcoptes scabiei is an ectoparasitic mite that causes the disease scabies and affects many species, including wildlife such as coyotes (*Canis latrans*) (Pence et al., 1983) and Chamois (*Rupicapra pyrenaica parva*) (Fernandez-Moran et al., 1997), as well as domesticated animals such as swine (Davis and Moon, 1990) and camels (*Camelus dromedarius*) (Nayel and Abu-Samra, 1986). Scabies has been implicated in greatly increasing mortality in wildlife species, often in spectacular epizootics (e.g. infected Chamois experienced 90% mortality rates, Fernandez-Moran et al., 1997; population structure of Red Foxes (*Vulpes vulpes*) in Northern Europe were strongly linked to the presence of scabies, Forchhammer and Asferg, 2000). In other populations, however, scabies had little effect on population size (Pence et al., 1983). Further, prevalence is often extremely high (e.g. 69% in coyotes, Pence et al., 1983). In domesticated animals, scabies can negatively influence condition, feeding efficiency, and reproduction (e.g. Davis and Moon, 1990). Thus, scabies can have severe conservation as well as economic consequences.

Although scabies is clearly an important disease, little has been done to examine its ecological epidemiology (but see Forchhammer and Asferg, 2000 for an interesting analysis of its dynamic impact on the

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host). Much work has concentrated on characterizing the clinical manifestation of scabies (e.g. Dahl, 1983), or the effectiveness of drug treatments on combating scabies in domesticated animals and humans (e.g. Clarke et al., 1992). Still other work has examined the biology of *S. scabiei*, examining the development of secondary host resistance (Arlian et al., 1994), and its survival (Arlian, 1989).

One reason for the paucity of studies on the epidemiology of scabies may be that it takes a tremendous amount of work to collect data sets of adequate size to allow meaningful analysis. Some studies have collected data on scabies prevalence (e.g. camels: Nayel and Abu-Samra, 1986; humans: Christophersen, 1978). One problem with prevalence data is that they are often incomplete, with either missing years or changes in collection of data/treatments. Perhaps more importantly, detailed information about the host populations is typically lacking. One exception, and the focus of this paper, is the coyote population in South Western Texas (Pence and Windberg, 1994). The researchers studying this population have collected information-not only on scabies prevalence but also on covote age structure, reproductive output, territoriality, population size indices, and other relevant population parameters. Despite the many remaining gaps in knowledge, there are enough empirical data to construct an exploratory model on the epidemiology of scabies in the context of host demographics.

In this paper, we develop a spatially explicit stochastic model that simulates the spread of a scabies epidemic in a coyote population. There are also a number of specific objectives. One is to synthesize the available data into a form that can be used in a population model. Another is to attempt to replicate epidemiological patterns and coyote demographic patterns. We want to determine whether our knowledge of processes underlying coyote demography and scabies biology is sufficient to approximate the empirical patterns observed.

2. Methods

2.1. Epidemiological and demographical patterns: a description

The epidemiological pattern of scabies in coyotes in South Western Texas to be analysed was as follows. After 1976, when the measured scabies prevalence was 15%, there was a lag period until 1978. Suddenly, the prevalence exploded to the peak phase of the epidemic, where 65-70% of individuals were infected (1979 and 1980). After 1980, prevalence declined to reach <20% by 1988 (Pence and Windberg, 1994).

We did not have actual population estimates, but instead had to rely on a relative population index. However, this was sufficient to show population changes over time. The empirical population index fluctuated greatly, but generally showed a slight decrease to 1981 and then increased again (data from Windberg, 1995).

2.2. Factors important for epidemiological patterns

At the simplest level, epidemics may be viewed as rates of gain and loss of infected and uninfected individuals. Generally, the gain of infected individuals may be affected by the transmission probability and also by the probability of encounter (Grenfell and Dobson, 1995). Transmission probability may also be related to time since infection. Parasites sometimes only become infective after a period (termed the latency period). The probability of encounter may be influenced by spatial processes and by host densities. Spatial processes include such factors as territoriality, visitation of neighbouring territories, and dispersal (Barlow, 1995). Additional processes include behavioural modification. Specifically, infected individuals may act, or be treated differently than uninfected individuals.

In general, the rate of loss is influenced by mortality and by rates of recovery. Natural rates of host mortality are relevant as these may both cause a reduction directly in infected individuals as well as influencing host densities. Infected individuals often have a higher probability of mortality. This is certainly true for scabies. For instance, for the coyote population studied, survival of adults was reduced from an average of 69% to an average of 23% (Pence et al., 1983). In scabies, mortality also is dependent upon age and social status (i.e. territorial versus transient individuals, see below). Rates of recovery may be influenced by genetic pre-dispositions (i.e. genetic resistance), or by experience. For example, canines appear to develop some secondary resistance, whereby the intensity of a scabies infection is much milder and short-lived in previously challenged individuals (Arlian et al., 1996). However, secondary resistance is not universal among species, or even within species in the same population. For instance, Little et al. (1998) found that red foxes did not develop any secondary immunity in contrast to previous work on domesticated canines (Arlian et al., 1996). Thus, it is unclear whether resistance existed within the coyote population. Further, if individuals differ in their ability to respond to mites (as appears to be the case), this difference may be genetic in origin, and may be subject to selective pressures. Thus, evolution (defined as changes in the frequency of alleles within a population across generations) may be a factor in the epidemiology of scabies.

Next, we provide a brief review of the natural history of coyotes and scabies to identify which processes would be included in the model.

2.3. Coyote biology and parameter estimations

2.3.1. Spatial organization

A coyote population comprises territorial and transient animals (Windberg and Knowlton, 1988). Territorial animals spend the majority of their time within their own territories, whereas transient animals spend much of their time within interstitial spaces between territories of other coyotes. Transients' range sizes are larger than range sizes of territorial animals. This spatial organization of coyotes is potentially important for scabies epidemiology because it affects which hosts contact one another and how often they do so. Further, territorial and transient animals differ greatly in their reproductive output and in their probabilities of survival.

Empirical estimates suggest that there were 1.3 territorial coyotes/km² and 0.7 transient coyotes/km². Further, the average territory size was 2.4 km², and the average range size of transients was 12.4 km² (Windberg and Knowlton, 1988) (see Table 1 for parameters and empirical estimates used in the model). Territories typically did not overlap, but were adjacent to one another. In contrast, transient ranges often overlapped several territories and ranges of other transient animals. Pence and Windberg (1994) have estimated that the core area of the epidemic covered 18,000 km² (the maximum range of the epidemic was 40,000 km²). We modelled only the core area in South Western Texas, as estimations of prevalence were with respect to this area (prevalence was quite low

at the outer edges of the epidemic). Thus, our initial population size was 36,000 coyotes (an overall density of 2 coyotes/km²). We used the average territory size (2.4 km^2) to construct a grid of 87×87 territories. For simplicity, we used square territories. Thus, each territory could be adjacent to eight other neighbouring territories. To generate transient ranges, we used the average transient range size (12.4 km^2) . We superimposed transient ranges to overlap one another.

2.3.2. Reproduction and population regulation

We filled territories with one alpha male and alpha female (we assumed only a single alpha pair within a territory). Empirical evidence suggests that only territorial alpha animals reproduce, having average 3.2 pups per reproducing female per year (Windberg, 1995). Only 39% of females reproduce in a given year. Thus, we calculated the number of litters per year as 1 female/km² × 18,000 km² × 0.39 litters/female. The number of litters could be less, if territories were empty and did not contain an alpha pair. If there were sufficient territorial pairs, we assumed this number of litters would be produced yearly.

Initially, we distributed transients randomly within our landscape. New transients were generated by the dispersal of non-dominant individuals. We assumed that dispersal occurred during the month of November [Windberg et al. (1985) found most instances of dispersal occurred between October and December]. Further, dispersal distance was on average 43 km (Windberg et al., 1985). We randomly generated dispersal distances (D) from a negative exponential probability density function (pdf = re^{-rD}), where r was the dispersal constant, chosen such that the average dispersal distance would be 43 km (i.e. r = 1/43). We modelled a closed system such that individuals reaching the edge of the range would turn back and continue the remainder of their dispersal in the opposite direction. This was needed to prevent individuals from accumulating at the edges of the spatial grid.

Unfortunately, there were no direct estimates of dispersal of juveniles or yearlings from territories. However, for a given year, we compared the expected number of territorial animals (number of existing territorial animals and the estimated number of offspring per year) to empirical estimates of the density of territorial animals (1.3 animals/km²). We simulated

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Table 1

Parameters used in the model and values obtained from empirical estimates

Parameter	Symbol	Value/equation 2.4 km ² 12.4 km ²	
Average territory size	NA		
Average transient size	NA		
Birth month	NA	4 (April)	
Dispersal constant	r	1/43	
Dispersal distance	D	$pdf = re^{-rD}$ (average $D = 43 \text{ km}$)	
Dispersal month	NA	11 (November)	
Infection constant	α	Fitted	
Initial proportion with resistance allele	γ	Fitted	
Juvenile annual survival	NA	42%	
Latency period	NA	1 month	
No. of pups per reproducing female	NA	3.2 pups	
Probability infecting another coyote	Ι	Eq. (4)	
Proportion females with pups	NA	0.39	
Proportion time in interstitial space	$T_{\rm i}$	0.05	
Proportion time in neighbor's boundary	$T_{\rm nb}$	0.04	
Proportion time in neighbor's core	$T_{\rm nc}$	0.02	
Proportion time in territory boundary	$T_{\rm b}$	0.10	
Proportion time in territory core	$T_{\rm c}$	0.79	
Proportion transient's time in a territory boundary	$T'_{\rm b}$	0.39	
Proportion transient's time in a territory core	T'_{c}	0.26	
Proportion transient's time in interstitial space	T'_i	0.35	
Recovery time for coyotes with resistance allele	λ	Fitted	
Relative proportion reproduction (infected vs. uninfected)	0	0.586	
Relative proportion time transient and territorial animal in same place	$P_{t't}$	Eq. (2)	
Relative proportion time two territorial animals in same place	P_{tt}	Eq. (1)	
Relative proportion time two transient animals in same place	$P_{t't'}$	Eq. (3)	
Survival: adult territorial	NA	82%/year	
Survival: infected adult	NA	42%/year	
Survival: infected juvenile	NA	9%/year	
Survival: transient	NA	39%/year	
Total area	NA	18,000 km ²	

Note that for survival of infected individuals, the upper empirical confidence limits were used (Pence et al., 1983). Further, an even higher juvenile survival was examined to improve the fit of the model, as a post-hoc hypothesis. Descriptions of the parameters and their use are given in the text.

juvenile dispersal until the expected density of territorial animals would be equal to or less than 1.3/km² in the following year. This also provided a regulatory mechanism within the population, as transient animals suffered much greater mortality rates. Birth occurred in spring (we modelled births as occurring in April, because Windberg (1995) indicated that dates of conception ranged from January to March, with 2 months of gestation). Thus, the population size would quickly expand and then decline over the remainder of the year.

We also modelled re-colonisation of empty territories (i.e. where either the alpha male or female had died). We allowed any transient individual whose range overlapped the empty territory and any local subdominant to re-colonise. These individuals had to have the same sex as the missing alpha animal.

2.3.3. Host survival

The probability of survival was dependent upon both social status (territorial or transient) and age (Windberg, 1995). Juveniles had a 42% yearly survival rate. One estimate of adult yearly survival was 69%. Another suggests that yearlings have lower survival than adults. These estimates grouped transient and territorial animals together, even though transients have 1.4–2.8 times greater chance of mortality. We used an empirical estimate of 82% survival for territorial adults, and assumed that transients had a 2.1 times greater probability of mortality than adults (i.e. the mid point between 1.4 and 2.8). The lower yearling survival compared with older adults was implicit in the greater proportion of transient yearlings.

2.4. Parasite-host dynamics

2.4.1. Encounter and transmission rates

Encounters occurred between overlapping or adjacent ranges, and were dependent upon the amount of time spent in the same location. There were no explicit empirical estimates of host encounter or of transmissions during a host encounter. However, territorial animals spent 79% of their time in their own core territories, 10% in the boundaries of their territories, 5% in interstitial spaces, 4% in the boundaries of other coyote territories, and 2% in the core territories of other coyotes. Transient animals spent 35% in interstitial spaces, 39% in boundary areas of other coyotes' territories, and 26% of the time in the core area of other coyotes (Windberg and Knowlton, 1988).

We assumed that the probability of encounters was linearly related to the proportion of time coyotes spend in the same area. For territorial coyotes, we considered neighbours to include all adjacent territories (there could be a maximum of eight neighbours). While it is possible that territorial coyotes also spend time in more distant territories, this would have likely been unimportant given the total time spent in neighbouring territories was only 6%. Territorial animals could also encounter one another in interstitial spaces. Thus, *P*_{tt}, the proportion of time two coyotes from neighbouring territories spent in the same area was:

$$P_{\rm tt} = \frac{2(T_{\rm c}T_{\rm nc} + T_{\rm b}T_{\rm nb})}{N_{\rm n}} + \frac{T_{\rm i}^2}{N_{\rm i}^2}.$$
 (1)

 T_c , T_{nc} , T_b , T_{nb} , T_i are the proportions of time spent in their own core territory, in the neighbour's core territory, in their own boundary, in the neighbour's boundary, and in interstitial spaces, respectively. N_n and N_i are the number of territorial neighbours and the number of interstitial spaces, respectively. N_n is 8, and N_i is 4. N_i corresponds to the four sides of a given territory. Thus, we only considered possible encounters within interstitial spaces directly between two territories (N_i is squared because that is the probability of both territorial neighbours being in the same interstitial space at the same time). N_i is a simplification, as diagonal neighbours would not have adjacent interstitial spaces.

The proportion of time transient and territorial animals spent in the same area $(P_{t't})$ was:

$$P_{t't} = \frac{(T_c T'_c + T_b T'_b)}{N_{t't}} + \frac{T_i T'_i}{N'_i}$$
(2)

where T_c , T_b and T_i were as above. T'_c , T'_b , T'_i were the proportions of time a transient spends in a territorial core area, territorial boundary area, and in interstitial spaces. $N_{t't}$ was the number of territories with which the transients' range overlaps ($12.4 \text{ km}^2/2.4 \text{ km}^2$). N'_i was the number of interstitial spaces with which the transients' range overlaps. We did not consider encounters between transients and territorial animals visiting other territories, as the probabilities would be negligible. Note that given relevant values and Eqs. (1) and (2), the majority of encounters involved transients ($P_{tt} = 0.005$ versus $P_{t't} = 0.06$). Thus, transients were the major vector of scabies transmission.

For possible transient-transient encounters, we calculated the overlapping territories and interstitial spaces of two transient ranges.

$$P_{t't'} = \frac{(T_{\rm c}^{\prime 2} + T_{\rm b}^{\prime 2})N_{\rm to}}{N_{t't}^2} + \frac{T_{\rm i}^{\prime 2}N_{\rm ti}}{N_{i'}^{\prime 2}}$$
(3)

where T'_{c} , T'_{b} , N'_{i} , $N_{t't}$ and T'_{i} were defined above. $P_{t't'}$ was the proportion of time two transients with overlapping ranges were in the same area. N_{to} and N_{ti} were the number of territories and interstitial spaces common to the ranges of both transients, respectively.

We assumed that transmission probability was linearly related to the proportion of two coyotes spent in the same area at the same time:

$$I = \alpha P \tag{4}$$

where *I* was the probability of infecting another coyote (maximum of one), α was a constant, and *P* was the proportion of time two coyotes spend in the same area. *P* could refer either to *P*_{tt}, *P*_{t't}, or *P*_{t't'} from Eqs. (1) to (3). α was a fitted parameter.

We further assumed that if a territorial coyote became infected, other coyotes living in the same territory would also become infected. Empirical evidence suggests that scabies is passed most frequently via close personal contact and that entire families are typically infected. We assumed encounters between territorial animals and strangers occurred via the adults, and pups did not encounter outsiders. Each potential infected/uninfected pair was tested for transmission using the probability from Eq. (4). Newly infected individuals did not become infectious until the following time interval (1 month), simulating a latency period (see Section 2.4.3).

2.4.2. Survival and reproduction

Infection status influenced the probability of survival. Adult yearly survival was reduced to 23% (confidence limits 5–42%). Juvenile yearly survival was reduced to 0% (confidence limits 0–9%) (Pence et al., 1983). Because juvenile yearly survival was based on a small sample size (six individuals) and a 0% survival seemed unlikely and would cause instantaneous death, we used the upper confidence limits.

Scabies also influenced reproduction by reducing the percentage of females with viable foetuses, depending upon the intensity of infection (Pence and Windberg, 1994). We approximated the average effect of infection by scabies on reproductive output by average reduction in viable foetuses, corrected for the proportion of individuals in the infection classes.

$$O = 1 - \frac{(p_a(o_c - o_a) + p_b(o_c - o_b))}{o_c}$$
(5)

or more simply,

$$O = \frac{p_a o_a + p_b o_b}{o_c}$$

where O was the relative proportion of infected compared to uninfected females that reproduce, p was the proportion of individuals in an infection class, and o was the proportion of females with viable foetuses. The subscripts *a*, *b*, and *c* were the infection classes: severe infection (>50% of body affected by mange), low and intermediate infection (<50%), and no infection, respectively. The proportion of individuals in each infection class changed over the epidemic. We used empirical estimates from the stationary (peak) phase of the epidemic (58% with low/intermediate infection, and 42% with severe infection). We reasoned that the peak would be the most crucial time to consider because it had the highest prevalence. We also only considered the individuals in spring, as this was when reproduction occurred. We used the adult estimates (excluding yearlings since most reproduction is by older adults) of percent females with viable foetuses, with 30, 48, and 69% for severe infection, low/intermediate infection, and no infection, respectively. This yielded an average estimate of O = 0.586, indicating that infected individuals had litters only 58.6% as often as uninfected individuals. We reduced the probability of infected individuals producing litters accordingly.

2.4.3. Parasitism and resistance

The progress of a disease within an individual can be separated into initial infection, latency period, infectious period, and recovery. We used 1 month as our latency period; this was when symptoms began to appear (e.g. humans, Arlian, 1989; foxes, Bornstein et al., 1995). We assumed that the probability of transmission was constant during the infectious period due to data limitations, although the probability of transmission should relate to mite load.

We also modelled recovery and genetic resistance. Some hosts appear to be able to respond, at least in part, to challenge by scabies (Arlian et al., 1994), whereas others do not (Little et al., 1998). Evidence from humans indicate mite numbers on individuals initially increase but eventually decline (Mellanby, 1972). Further, it seems likely that individuals differ genetically in their susceptibility to scabies and, therefore, that selection for resistance to scabies may be possible. Development of secondary resistance (acquired immunity) appears to be heterogeneous, suggesting the potential for inherent differences among individuals (e.g. only 60% of individuals appear to show secondary resistance). Further, some individuals appear to be naturally immune to scabies, and to spontaneously recover (Arlian et al., 1994).

We did not have explicit data on the genetics of resistance nor its possible side effects. Consequently, we modelled the simplest genetic system. We assumed that the resistance allele was neutral in the absence of scabies and we fit the proportion of the resistance allele at the beginning of the epidemic (γ). In the presence of scabies, individuals with the resistance allele recovered after λ time intervals (months), whereas those without the allele did not recover. Offspring could inherit alleles from their parents, simulating natural selection. We examined dominant, co-dominant, and recessive resistance alleles. These generated similar epidemiological/demographic patterns, although the fitted values of γ and λ necessarily differed between models. Thus, we only presented results for dominance. We note, though, that it is unknown whether, or to what degree, coyotes show such resistance. As such, our initial simulations did not include resistance.

2.5. Simulations and fitting the data

We simulated the scabies epidemic using discrete time steps with time intervals of one month. The order of simulated processes was as follows: death, birth (only in April), recolonisation, dispersal (only in November), and infections.

We used least squares to measure the goodness of fit of the model to the data, for both prevalence and population size. To integrate this information into a single metric, we first standardized the least squares by the variation in the observed variable, such that contributions from prevalence and population size would be comparable. For prevalence:

$$LS_{prev} = \frac{\sum (y_i - x_i)^2}{\sum (y_i - y_m)^2}$$
(7)

where y was the observed value, y_m was the mean observed value, and x was the simulated value. For population size, we only had a relative index and could only examine population trends. For population size we used regression techniques to determine the best fitting relation between simulated population size and population index:

$$LS_{pop} = \frac{\sum (y_{i} - \beta x_{i} - \iota)^{2}}{\sum (y_{i} - y_{m})^{2}}$$
(8)

where β and ι were fitted parameters from regression. Note that $1 - LS_{pop}$ is the coefficient of determination from linear regression. The integrated metric of fit was simply $LS_{tot} = LS_{prev} + LS_{pop}$. For simulated population trends in the wrong direction ($\beta < 0$), we included a penalty ($LS_{pop} = LS_{pop} + 1000$) such that, if they were generated, population trends in the correct direction would be preferred.

To determine the best fitting parameters (i.e. that resulted in the lowest LS_{tot}), we used a linear search for the optimal strength of resistance (λ), because λ was an integer (simulated $\lambda = 1-10$ months). At each λ , we used the simplex algorithm (Press et al., 1995) to find the optimal probability of transmission (α) and proportion of hosts resistant (γ) , which were continuous. We began the procedure at a central point ($\alpha =$ 0.4 and $\gamma = 0.4$ for one vertex and randomly generated other vertices) and used a penalty (LS_{tot} = 10,000) for invalid parameter values ($\alpha < 0$ and $\gamma < 1$ 0). For our stopping rule, we ended the simplex procedure when the maximum difference in parameter values between vertices converged to less than 0.01. Because our simulations had a stochastic component (and therefore, LStot was variable for each parameter set), we refit our simulations. We re-ran this simplex procedure 99 times at the optimal value of λ and the two adjacent λ values (i.e. 33 times for each λ value) to examine the variability in best-fit (LS) values.

3. Results

It was not possible to get good fits simultaneously to prevalence and population trends using the basic model. The observed prevalence pattern could be generated but the population did not recover (Fig. 1). We therefore considered additional processes that could allow population recovery.

One potential mechanism was to allow longer distance re-colonisation, rather than limiting re-colonibreak sation to transients whose range overlapped the empty territory. We permitted uninfected nondominant or transient individuals to colonize empty territories anywhere in the simulated landscape. This could occur if coyotes occasionally conduct long distance exploration (Windberg and Knowlton, 1988). Here, coyotes spend most of their time within defined ranges, but occasionally explore much further distances. Because such occurrences are rare, it might not directly affect transmission probabilities but would allow empty territories to become occupied. We also considered the possibility that infected animals were less likely to colonize empty territories, either because of morbidity or reduced condition/competitive ability (Pence et al., 1983; Mörner, 1992). In this scenario, infected animals colonized territories only when non-infected animals were not available. Although this model permitted the population to recover, recovery was still too slow. Thus, this model still could not reproduce the observed patterns.



Fig. 1. Graph showing the best fit of the model to the empirical prevalence data, fitting the transmission constant, α . This model was our "basic" model, where host evolution was not considered and empty territories could only be re-colonised by local coyotes. Although prevalence could be fit, the coyote population typically crashed. Prevalence and population size could not be fit simultaneously using this model. Top panel shows prevalence and bottom panel shows population size. The left *y*-axis refers to simulated population size, and the right *y*-axis refers to the empirical population index. Error bars represent one standard deviation.

Next, we hypothesized that inherited host resistance might permit a faster recovery period. Using this model, we could fit the observations much better (Fig. 2, Table 2). The timing of the simulated epidemic was reasonable. The peak phase occurred in 1979 and 1980, as observed. The level at the end of the declining phase was similar to empirical estimates (i.e. ca. 15%). However, the model under-estimated the magnitude of the peak by almost 25%. Further, the prevalence declined more sharply than the empirical estimates. In contrast to the previous two models, the population demographics followed the empirical pattern, with a minimum in 1981, and reasonable population recovery after. Further, this suggests a 1-2-year-lag from the peak prevalence to minimum population size.

Next, we hypothesized that we might increase the prevalence by increasing the survival of infected individuals. To examine this, we chose to increase the survival of infected juveniles, because the empirical estimate of survival was based on a sample size of six individuals, and might therefore have had a large degree of uncertainty associated with it. We doubled the survival probability of infected juveniles. We found that the epidemic reached higher peak prevalence at the appropriate time, and declined at a more reasonable rate as well. Overall, it reduced the summed squared errors by 17% (Fig. 3, Table 2).

The epidemiological pattern was largely driven by the evolution of resistance. In our model, the proportion of resistant alleles increased from an initial level



Fig. 2. Graph showing the fit of the model to the empirical data, using the best fitting sets of parameters (α , λ , and γ), fitted simultaneously to prevalence (top panel) and population size (bottom panel). In contrast to Fig. 1, host resistance was allowed to evolve and re-colonisation could occur by any transient or non-dominant uninfected individual. Values are based on simplex fits of α and γ repeated a total of 99 times across the highest three values of λ (a discrete parameter). The left y-axis refers to simulated population size, and the right y-axis refers to the empirical population index. Error bars represent one standard deviation.

Table 2 The best fits for two models, (A) only heritable resistance simulated and (B) heritable resistance and high juvenile survival simulated

λ	A			В		
	2	3	4	1	2	3
γ	0.16 (0.049)	0.19 (0.014)	0.20 (0.018)	0.090 (0.011)	0.13 (0.011)	0.15 (0.011)
α	0.50 (0.050)	0.45 (0.014)	0.39 (0.018)	0.30 (0.014)	0.38 (0.011)	0.31 (0.011)
LStot	1.43 (0.087)	1.42 (0.012)	1.44 (0.013)	1.22 (0.023)	1.17 (0.013)	1.18 (0.012)
LSprev	0.63 (0.071)	0.58 (0.018)	0.55 (0.018)	0.33 (0.016)	0.33 (0.020)	0.28 (0.011)
LSpop	0.80 (0.024)	0.84 (0.015)	0.89 (0.015)	0.88 (0.024)	0.84 (0.022)	0.90 (0.011)

Simplex fitting procedures were repeated 99 times for each model to examine variability due to stochasticity. The optimal average fits (minimum LS_{tot}) occurred when $\lambda = 3$ for A and $\lambda = 2$ for B. We examined adjacent λ values to examine variability (33 simplex simulations at each λ value). LS_{tot} values largely overlapped between λ values. Although resistance ($\lambda > 0$ and $\gamma > 0$) was necessary to generate reasonable patterns, LS_{tot} values were relatively insensitive to the exact values of λ and γ . In contrast, high juvenile survival improved the fit, reducing total squared errors by 17% ($1 - LS_{tot}B/LS_{tot}A$). λ was the effect of resistance (months to recovery), γ was the initial proportion of resistant alleles in the population, and α was the transmission constant. LS_{tot} , LS_{prev} , and LS_{pop} were the total least squares, and the least squares contributions from fits of prevalence and population size, respectively. Standard deviations are in parentheses.



Fig. 3. Graph showing the fit of the model to the empirical data, using the best fitting sets of parameters (α , λ , and γ), fitted simultaneously to prevalence (top panel) and population size (bottom panel). Modelled processes were the same as in Fig. 2, except that survival of infected juveniles was increased to 0.2. Values are based on simplex fits of α and γ repeated 99 times across the highest three values of λ . The left *y*-axis refers to simulated population size, and the right *y*-axis refers to the empirical population index. Error bars represent one standard deviation.

of 13–60% over 12 years (for $\lambda = 2$, $\alpha = 0.38$, $\gamma = 0.13$).

3.1. Other demographic factors

We also had data on other population demographics. For instance, empirical estimates suggested that there was an overall population density of about 2 individuals/km² and that territorial animals accounted for approximately 64% of the population, and that on average juveniles accounted for 33% of the total number of individuals (Windberg, 1995). In the absence of scabies and measuring throughout the year, we obtained estimates of 2.1 individuals/km², 64.1% territorial animals, and 30% juveniles. However, in the presence of scabies, our estimates were 1.6 individuals/km², 78% territorial animals, and 33% juveniles.

4. Discussion

The model suggests that long distance exploration was very important for population stability. Without this, Allee effects occurred. Specifically, when we modelled only local re-colonisation, there was a threshold, below which the population quickly went to extinction. Here, scabies caused high mortality of individuals causing territories to be left empty and the resulting decrease in reproduction, which in turn reduced the population size further in the subsequent generation, until population extinction occurred. Exploration appears to be a reasonable mechanism for coyote population stability necessary for the patterns observed, as long-range explorations could occur 5–6 times per month (Windberg and Knowlton, 1988).

The evolution of resistance was also important for population recovery, for the declining phase of the epidemic, and for scabies prevalence to remain low indefinitely. In the absence of such resistance, both population size and scabies prevalence would form a long-term cyclical pattern. Thus, some process that permits reduction in susceptibility to scabies across generations was necessary to explain the patterns observed. The model demonstrated that host evolution was a logically feasible mechanism of the population recovery, which could operate under realistic parameter values even within the short time frame of the epidemic, and could largely explain the patterns observed. Further, this would be consistent with previous scabies epidemics. The last scabies epidemic was in the late 1920s, lasting for approximately 10 years, and persisting at very low prevalence for the next 50 years (<1% prevalence, Pence et al., 1983). Increased genetic resistance is one possible explanation for the long period between epidemics. The introduction of a new scabies strain or a mutation in the strain may explain the 1976 epidemic. Differences in evolution and re-colonisation may result in the differing consequences of scabies between populations and between species.

Unfortunately, the empirical data simply do not exist to test these possibilities as a priori hypotheses. However, we note that in the absence of these processes or processes with similar effects—the existing empirical population demographic estimates should result in a discrepancy between prediction and observations (i.e. the numbers do not add up). Thus, some additional process or modification from the strict empirical estimates is required.

While the general fit of the model was fairly good, minor discrepancies existed between the model output and the empirical results. The estimate of population density and structure was good in the absence of scabies. However, in the presence of scabies, the model predicted a lower population density and a higher proportion of territorial animals. Further, researchers have suggested that juveniles do not typically disperse in this population but rather delay their dispersal (Andelt, 1985). However, when we modelled delayed dispersal, the proportion of territorials was even higher (84%), the population density was overestimated (2.25 individuals/km²), and the fit to the epidemiological and demographic data was consistently poorer (simulations not shown).

It may be that age determination in coyotes has some error associated with it, and that juveniles disperse at least under some conditions. For instance, Windberg et al. (1985) recovered half the female juveniles dead 20–80 km from their marking locations, and suggested that this was due to egress of juveniles. In any case, these discrepancies highlight aspects of coyote biology that require further attention and explanation.

Other inferences that may be drawn from this model include the simulated observation that evolution even of large mammals may occur within relatively short ecological time frames, and should not be discounted in models predicting the evolution of parasite virulence (e.g. May and Nowak, 1995; Leung and Forbes, 1998). Also, population sizes were relatively stable, despite high prevalence and high mortality of infected individuals. Thus, models that assume a constant population size may be biologically reasonable under some realistic scenarios. While this model demonstrates that these processes and patterns are logically biologically plausible, they remain speculative and in need of direct tests.

Other modelling approaches, such as a modified SEIR model or another deterministic formulation, may be possible and would be worth exploring. Originally, we had considered a standard SEIR approach and a non-spatial simulation approach (data not shown). However, both of these were not sufficient the standard SEIR was able to capture the simple epidemic pattern, but was not able to capture both epidemic and demographic patterns simultaneously; the non-spatial model could not capture the timing of the epidemic in that the prevalence increased too quickly. Thus, modifications would be necessary. Here, we used a spatially explicit, stochastic, individual based simulation model. The results of this manuscript suggest several processes could be important and should be considered regardless of modelling approach. Any formulation should be spatially explicit, given the importance of the dispersal kernel. Further, we should consider population structure, especially genetic classes (resistant homozygous, not resistant homozygous, heterozygous) given the suggestive importance of evolution to long term population recovery, and social classes (territorial and transient) given their difference in transmission probabilities and survival.

5. Conclusions: implications for field studies

The original formulation, based on empirical estimates of population demographic processes, was insufficient to capture the empirical patterns. We required several novel hypotheses. We demonstrated that, theoretically, rare long distance dispersal/recolonisation was important for recovery of the population in the presence of a strong disease epidemic (due to an Allee effect). Further, host resistance could evolve, and was an important contributor to the parasite-host dynamics observed. In addition, the trends were much more reasonable when dispersal could occur during the juvenile phase. We should recognize, however, that these hypotheses are untested and require empirical validation to see whether they occur in this system, and whether they might be important in other systems.

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