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LOCALIZED DEER ABSENCE LEADS TO TICK AMPLIFICATION

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Abstract. Deer support high tick intensities, perpetuating tick populations, but they do not support tick-borne pathogen transmission, so are dilution hosts. We test the hypothesis that absence of deer (loss of a dilution host) will result in either an increase or a reduction in tick density, and that the outcome is scale dependent. We use a complementary methodological approach starting with meta-analysis, followed up by a field experiment. Meta-analysis indicated that larger deer exclosures reduce questing (host-seeking) tick density, but as the exclosure becomes smaller (<2.5 ha) the questing tick density is increased (amplified). To determine the consequences for tick-borne pathogen transmission we carried out a field experiment, comparing the intensity of ticks that fed on hosts competent for tick-borne pathogen transmission (rodents) in two small (<1 ha) deer exclosures and their replicated controls. Intensity of larval ticks on rodents was not significantly different between treatments, but nymph intensity, the tick stage responsible for tick-borne encephalitis (TBE) transmission, was higher in deer exclosures. TBE seropositive rodents were found in a deer exclosure but not in the controls. We propose that localized absence of deer (loss of a dilution host) increases tick feeding on rodents, leading to the potential for tick-borne disease hotspots.

Key words: deer exclosures; dilution effect; disease control; hotspots; rodents; TBE; tick amplification; tick-borne disease; tick reduction; ticks.

Introduction

At the global scale the majority of vector-borne diseases exhibit a broad geographical distribution while at the local scale many occur in distinct geographical hotspots. Vector distribution can be predicted from broad climatic variables (Randolph et al. 1999), but vector-borne disease hotspots (foci of infection) tend to occur at a fine scale, determined in part by the spatial distribution of hosts competent for tick-borne pathogen transmission, relative to the non-competent host species (Van Buskirk and Ostfeld 1995, Zeman and Daniel 1999, Ostfeld and Keesing 2000 *a*, *b*). The pathogens transmitted by ixodid ticks tend to be focal in their distribution and fit these criteria well, because each of the three tick stages utilizes multiple hosts of different species that differ in their tick-borne pathogen transmission competency and spatial distribution (Labuda et al. 1997, Gilbert et al. 2001).

The adult female tick takes its final blood meal from a large vertebrate host, typically deer, and so one form of tick and tick-borne disease control has been to exclude deer from defined areas (Bloemer et al. 1986, Wilson et al. 1988, Stafford 1993, Wilson 1998, Ginsberg and Zhioua 1999, Ginsberg et al. 2002). This method of disease control presumes that removal of the definitive tick host (the deer) will prevent the tick life cycle from being completed, thus leading to fewer ticks feeding on woodland rodents, which are the most competent host for a range of tick-borne pathogens. As such, deer are referred to as dilution hosts, dilution referring to the fact that the deer are not competent for tick-borne pathogen transmission and so are dead-end hosts. This relationship has been incorporated into mathematical models of tick-borne disease control, which predict that absence of deer will prevent the recruitment of tick larvae. This reduction in tick density in the environment will then lead to reduction or elimination of tick feeding on rodents and so ultimately lead to tick-borne disease fade out (Norman et al. 1999, Gilbert et al. 2001, Rosa et al. 2003). We term the reduction in questing (host-seeking) ticks and/or tick feeding on reservoir competent hosts the tick reduction hypothesis. The mathematical models described previously do not take account of spatial scale, but yet the empirical evidence for tick reduction via deer exclusion appears equivocal. Deer exclusion sometimes results in high densities of questing ticks available in the exclosure, as evidenced from dragging studies (sampling of ticks in the environment), especially when the exclosure size is small (Daniels and Fish 1995, Ginsberg et al. 2002). The consequence of increased tick availability in the environment is the potential for increased tick feeding on rodents thereby increasing tick-borne pathogen prevalence (Ostfeld and Keesing 2000*a*, *b*, Gilbert et al. 2001). However, few studies have examined the consequences of deer exclusion for tick biting intensity on rodents (but see Deblinger et al. 1993,

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TABLE 1. Description of the data used for the meta-analyses.

Tick species and life stage	Exclosure size (ha)	Years after deer exclusion
Ixodes scapularis		
Adult	1	1
Adult	1	2
Nymph	1	1
Larva	3.5	7
Larva	3.5	8
Nymph	3.5	7
Nymph	3.5	8
Adult	3.5	7
Adult	3.5	8
Larva	7.4	1
Nymph	7.4	1
Adult	7.4	1
Amblyomma americanum		
Nymph	1	3
Nymph	1	4
Nymph	1	5
Nymph	1	6
Adult	1	2
Adult	1	3
Adult	2.43	1
Adult	2.43	2
Adult	2.43	4
Larva	2.43	4

Notes: All ticks included in this meta-analysis are three-host ticks; that is, during their development they feed on three different hosts. All studies estimated tick density using dragging techniques; this involves trailing a piece of material across vegetation for a distance or time interval. References for *I. scapularis* are Ginsberg and Zhioua (1999) for 1-ha exclosures and Stafford (1993) for 3.5-ha and 7.4 ha exclosures. References for *A. americanum* are Ginsberg et al. (2002) for 1-ha exclosures and Bloemer et al. (1986) for 2.43 exclosures.

Wilson et al. 1988). We refer to the increase of ticks in the environment, determined using dragging and/or an increase in feeding intensity of ticks on rodents, as the tick amplification hypothesis.

Therefore we propose the hypothesis that deer exclusion may result in either tick reduction and disease fade-out or tick amplification and disease hotspots and that the outcome will be scale dependent. To address this we undertook two studies. First, we quantitatively examined the effect of deer exclosure size on questing tick density using meta-analysis of published studies. Second, we tested empirically whether deer exclusion from <1-ha plots results in tick amplification or reduction on tick-borne pathogen transmission-competent rodents and the effect this has on tick-borne disease seroprevalence. We used the model system of woodland rodents and tick-borne encephalitis (TBE), a zoonotic pathogen endemic to mainland Europe.

METHODS

Meta-analysis

We compiled a data set of published information where workers had recorded questing tick densities in deer exclosures and their matched controls ranging in size from 1 to 7.4 ha. (Table 1). To reduce the number of covariates between different studies the criteria for inclusion in the meta-analysis were studies that had field sites based in North American woodland, sampled a three-stage tick, and used dragging as a tick density estimation method. These data were compiled from four different deer exclosure studies and consisted of 22 data points, where each data point relates to a different exclosure and different tick life stage sampled over time. As such, each data point is not truly independent and in meta-analytic terms constitutes a correlated design. To overcome the problems associated with repeated measures of ticks from the same exclosures we combined data points from each of the four different exclosure sizes (Table 1). Because the sample sizes for meta-analysis are small we treat these analyses as indicative of tick reduction or amplification, but test our hypothesis empirically with a field experiment.

We first calculated the effect size (E_S), for each data point. This is the average difference in questing tick density between exclosures and controls. A negative effect size indicates a decrease in ticks associated with deer exclusion (tick reduction), and a positive effect size indicates an increase in ticks associated with deer exclusion (tick amplification). The effect size was calculated using Hedge's d because it is not biased by unequal variances or small sample sizes (Rosenberg et al. 2000). We pooled data points to produce a mean effect size for each of the four different exclosure sizes. We estimated the effect size associated with deer exclosures that ranged in size from 1 to 7.4 ha using a fixed effect continuous meta-analytic model. The model output was bootstrapped and analysis was carried out in Metawin 2.0 (Rosenberg et al. 2000).

Empirical analyses

Study site and rodent sampling.—Longitudinal monitoring of woodland rodents was undertaken every fortnight for two trap nights between April and September 2000 (4992 trap nights) with Ugglan live traps (Grahnab, Sweden) using standardized techniques (Perkins et al. 2003). The study area was located in a TBE hotspot in the Italian Alps (Hudson et al. 2001). Exclosure fences were 2 m high (4 × 4 cm mesh) excluding both roe (*Capreolus capreolus*) and red deer (*Cervus elaphus*), but did not restrict the movement of woodland rodents: *Apodemus flavicollis*, yellow-necked mouse, and *Clethrionomys glareolus*, bank vole. The exclosures had been in place for 16 years prior to the start of the experiment to avoid any short term temporal effects associated with deer exclusion. We used two replicated deer exclosures with paired controls containing 64 traps, covering an area of 0.64 ha.

For each rodent capture we recorded larval intensity, nymphs plus adult female ticks, and co-feeding ticks (larvae–nymph aggregations; see Plate 1). TBE is transmitted nonsystemically between co-feeding ticks, and therefore co-feeding intensity provides an indication of TBE transmission. Other tick-borne pathogens, such as

PLATE 1. Ticks (*Ixodes ricinus*), including a co-feeding aggregation, on a yellow-necked mouse (*Apodemus flavicollis*). Photo credit: V. Tagliapietra.

Lyme, are usually transmitted systemically, and in terms of tick-borne pathogen transmission potential the nymph intensity is the important variable. Host biometrics recorded included sex and body mass. A tail-tip blood sample (20 μL) taken from each capture was tested for TBE antibodies, using a standard ELISA test. The antibody titre was compared to a TBE-free population of *A. sylvaticus*, from Ireland. An antibody dose unit level higher than the highest level observed in the TBE-free population ($n = 61$) was taken to be TBE seropositive.

Statistical analyses.—To determine the effect of deer exclusion on rodent tick intensity we used general linear mixed models (GLMMs) alternatively using larval, nymphs plus adult female ticks, co-feeding tick intensity (negative binomial errors), and finally TBE seroprevalence (binomial errors) as the response variables. GLMMs were used to overcome the problem of temporal autocorrelation associated with repeated tick measures and were carried out using IREML in Genstat 6 (Genstat 2002). Adult ticks were observed on rodents and so were summed with nymph intensity, because these tick stadia are potentially infected with TBE, to produce a "potentially infected ticks" variable.

To investigate the potential mechanisms that allowed tick persistence in the absence of deer we determined if there were spatial or ecological patterns in rodent tick intensity within the trapping grids. The exclosures covered an area of <1 ha, and with the mean home range of the rodents in this study estimated at 0.28 ha for both species (minimum polygon method, using trapping records), we expected some "fence crossers" to import ticks, which would create a positive gradient in tick intensity toward the edge of the exclosures. In contrast, control grids should show no obvious spatial patterns. We assigned geographic coordinates to each trap location linearly in the x and y direction and calculated the mean larval intensity and nymph plus adult tick intensity of all animals caught in each trap location. These were then used as the response variables with the traps' coordinates and distance from the center of the trapping grid as explanatory variables in a GLM with negative binomial errors (SPlus 2000). Ecologically we had previous empirical evidence to suppose that key hosts may account for the majority of tick intensity and so also TBE persistence (Perkins et al. 2003). Therefore we examined whether exclosures and controls exhibited differences in host sex, body mass, and density with deer exclusion as the response variable in a GLM (generalized linear model) with binomial errors (SPlus 2000).

RESULTS

Meta-analysis: the effect of deer exclusion on questing tick density

We found a significant negative relationship between exclosure size and effect size (slope = −0.2188, SE = +0.0582, $P < 0.001$; Fig. 1), supporting tick reduction in large exclosures and tick amplification in small exclosures (Fig. 1). An effect size of zero represents the null hypothesis of no difference in questing tick density between exclosures and controls, which occurs at 2.5 ha (Fig. 1). This is indicative that a threshold deer exclosure size exists where either tick amplification (<2.5 ha) or

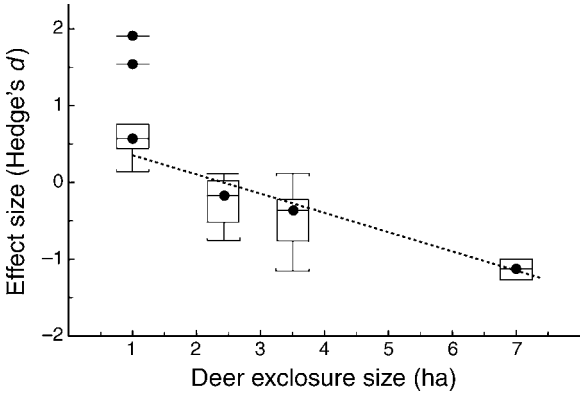

FIG. 1. Box plot and regression (dotted line) of the standardized mean difference (solid circle in box) in questing tick densities between the deer exclosures and controls (effect size) over a range of deer exclosure sizes. The boxes denote the 25th and 75th percentiles, with the whiskers (horizontal lines) showing 1.5 times the interquartile range. Outliers are shown as solid circles, with a single horizontal line. A positive effect size denotes an increase in tick density in deer exclosures relative to controls, and a negative effect size denotes the converse. Note that the effect size of zero is the null hypothesis of no difference in questing ticks between treatments and that it occurs at ~2.5 ha.

TABLE 2. Larval, nymph, adult, and co-feeding tick prevalence and geometric mean intensity, in <1-ha deer exclosures (E) and their matched controls (C).

	Range		Tick prevalence (%)		Tick intensity, geometric mean†		Total no. ticks counted	
Life stage	C	E	C	E	C	E	C	E
Larvae	0–63	0–86	93.7	90.0	9.45	8.18	2467	3416
Nymphs	0–4	0–7	17.8	29.2	0.16	0.34	45	117
Adults	0–1	0–1	1.2	2.9	0.02	0.02	2	6
Co-feeding	0–3	0–5	13.8	18.2	0.12	0.18	30	60

Notes: Range is the difference between the highest and lowest tick intensities found in the rodent population. Tick prevalence is the proportion of the host population infected with ticks over the course of the study. Tick intensity is the mean tick burden of both infected and uninfected rodents. Tick prevalence was calculated per capture. Note that the highest intensity of ticks was consistently observed in the deer exclosures.

† Using the equation $y = \text{mean}[\log_{10}(x + 1)]$, geometric mean $= (10^y) - 1$.

tick reduction (>2.5 ha) could occur. On the basis of these findings we set out to test empirically whether small exclosures (<1 ha) would result in tick amplification in the rodent population.

Empirical analysis: the effect of deer exclusion on rodent tick intensity

A total of 4992 trap nights resulted in 530 captures of 208 individual rodents. The number of yellow-necked mice in exclosures was higher ($n = 71$ mice) than in the controls ($n = 47$), while the numbers of bank voles were comparable (43 vs. 47 voles, respectively). Preliminary analyses revealed no significant differences in tick intensities between the two rodent species (negative binomial GLM; $\chi^2 = 0.97$, df = 1, $P = 0.323$), and so they were grouped for all analyses. All ticks on the rodents were identified as *Ixodes ricinus*.

We found no significant difference in host larval intensity between deer exclosures and control grids ($\chi^2 = 2.37$, df = 1, $P = 0.124$), although the mean tick intensities were higher in the deer exclosures (Table 2). Exclosures hosted significantly higher nymph plus adult female tick intensity ($\chi^2 = 7.49$, df = 1, $P = 0.006$) and co-feeding tick intensity ($\chi^2 = 6.13$, df = 1, $P = 0.013$), therefore supporting the tick amplification hypothesis at this small scale. In addition, TBE seroprevalence was significantly higher in the exclosures ($\chi^2 = 21.11$, df = 1, $P < 0.001$) compared with controls, although just one exclosure hosted all the TBE infected rodents. Although few in numbers, it is interesting to note that 2.9% of rodents in the deer exclosures hosted engorged adult ticks compared to just 1.2% of hosts in the control sites.

Empirical analysis: spatial and ecological differences between treatments

We carried out a spatial statistical model to determine if tick intensity of rodents increased or decreased toward the center of the deer exclosures, in comparison to the controls. We found that both larval and the nymph plus adult female tick intensity was highest in the center of the deer exclosures, declining significantly toward the edge (larvae, $\chi^2 = 5.99$, df = 1, $P = 0.014$; nymphs plus adult females, $\chi^2 = 24.68$, df = 1, $P = <0.001$). In contrast, ticks in the control sites showed no significant spatial patterns (larvae, $\chi^2 = 0.09$, df = 1, $P = 0.764$; nymphs plus adult females, $\chi^2 = 0.03$ df = 1, $P = 0.864$). We caught significantly more male rodents of high body mass in the exclosures (sex : body mass interaction, $\chi^2 = 4.39$, df = 1, $P = 0.036$), but found no significant differences in rodent density between treatments ($F = 0.46$, df = 1, 64, $P = 0.929$).

DISCUSSION

In this paper we examined whether loss of a dilution host by excluding deer, at different scales, would result in tick reduction or tick amplification and the consequences of this for tick-borne disease emergence. First, using meta-analysis we determined that if the deer exclosure was >2.5 ha, then a reduction in questing tick density occurred, and below this size, tick amplification occurred (Fig. 1). We then tested the consequences of this for tick biting intensity and tick-borne pathogen seroprevalence in a rodent population using replicated deer exclosures and controls <1 ha. Nymph plus adult female ticks and co-feeding tick intensity, but not larval intensity was significantly higher in deer exclosures than controls (Table 2). In addition 8% of the rodent population were seropositive to TBE in one of the deer exclosures while no animals were seropositive in the controls, providing evidence for tick amplification (i.e., high vector biting intensity and a TBE hotspot). Although TBE occurred in only one deer exclosure, the potential for TBE transmission was high in both exclosures, with significantly more nymphs plus adult female ticks and co-feedings occurring on rodents than in controls (Table 2).

Our meta-analysis is the first quantitative synthesis of previously published data so providing insight that is missed qualitatively and may be obscured by single studies using large deer exclosures (Wilson et al. 1988, Lane et al. 1991). However, a drawback to all meta-analysis is that of confounding variables, including ecological differences between populations in the different field sites. For example, the year in which tick sampling occurred after deer were removed varies between studies (Table 1), and one could argue that

this may bias results if the tick amplification effect is short-lived. Immediately after removal of deer the density of questing ticks is typically very high (e.g., Rand et al. 2004) because there are no longer deer to feed on. This may be expected to fall over time as the ticks fail to find blood meals due to the absence of deer. Unfortunately there are not enough data to examine this question with meta-analysis. However, the exclosures used in our empirical experiment had been established for over 16 years, and the tick amplification effect is still seen, suggesting that a temporal effect does not exist in small exclosures. We attempted to reduce confounding factors by including similar studies from North America. However, we believe further studies concentrating on the tick amplification hypothesis would provide important contributions to a more robust meta-analysis. Nonetheless, our meta-analysis provides some support for tick amplification at small spatial scales, which we corroborate with the empirical experiment and which provides a unique analysis of the effect of deer exclusion on tick-borne pathogen competent hosts.

One drawback to studies that estimate the efficiency of tick-borne disease control by collecting data on questing ticks only is that they may not reflect the true tick intensity (and pathogen transmission potential) on rodents. This mismatch between questing and biting ticks has been illustrated by Daniels et al. (1993) where questing ticks were fewer in exclosures compared to controls but the prevalence of Lyme infected nymphs did not differ, suggesting high tick intensity and tick-borne pathogen transmission on rodents in deer exclosures. In this respect, our field experiment was the essential component of this study because it allowed us to determine if pathogen transmission increased on rodents as a consequence of tick amplification.

Empirically, nymph plus adult female ticks and co-feeding tick intensity, but not larval intensity, on the rodents was significantly higher in deer exclosures than in controls (Table 2). The increased tick–host contact rate can increase tick-borne pathogen transmission and may explain the TBE hotspot observed in one of the deer exclosures. In support of tick-borne pathogen hotspots at small spatial scales Allan et al. (2003) found in fragmented forest patches (<2 ha) where an ixodes tick population was not produced by deer (therefore analogous with deer exclusion) that the density of nymphs infected with the spirochete that causes Lyme disease was extremely high and decreased exponentially with increasing patch size (up to ~ 8 ha). Indeed the density of infected nymphs in the very smallest forest fragments (~ 1 ha) was higher than any previously published infection levels. This, in combination with our study suggests alternative tick-borne pathogen transmission states exist at different spatial scales, a factor that should be incorporated into future multi-host models examining tick-borne disease persistence.

We propose that the mechanism causing tick amplification at small scales can be explained by a previously unrecognized spatial aspect to the dilution effect. Loss of a dilution host (deer) at a small spatial scale results in increased tick availability in the environment (indicated from meta-analyses) and increased tick intensity and tick-borne pathogen transmission on rodents (Ostfeld and Keesing 2000*a, b*). Empirically, we find evidence for this from observations of a negative gradient from the center to the edges of the exclosures in the intensity of larval and nymph plus adult female ticks feeding on rodents. We had expected this gradient to be positive due to rodents that live at the edges of the exclosures frequenting deer habitat and so picking up more ticks. Our observed negative gradient suggests that the reduced tick intensity of rodents at the edges of the exclosures is a function of "sharing" ticks with deer. However, we must ask "How is the tick population perpetuated in the exclosures when deer are lost"?

To a certain extent the life cycle must be perpetuated by rodents importing ticks, which explains why tick reduction occurs in larger deer exclosures, although this remains to be tested. However, several other possibilities may contribute to increased tick intensities in deer exclosures including the rodents maintaining the tick life cycle by feeding adult ticks, supported by the fact that 3% of rodent captures hosted adult ticks, a highly unusual observation. Plus, increased plant growth and mat layer may reduce tick desiccation leading to increased tick survival (Flowerdew and Ellwood 2001). In addition we observed a predominance of sexually mature male rodents in the deer exclosures, and putative optimum rodent conditions may have allowed males of good physical condition to support high parasite burdens.

From a practical viewpoint this study demonstrates that deer exclusion as a tool for tick reduction and tick borne disease elimination works at large spatial scales; however, it is likely to amplify ticks and produce tick-borne disease hotspots at small spatial scales. Therefore we may expect a tick-borne disease hotspot to occur where deer are locally absent, but rodents remain ubiquitous, for example, fragmented forest patches (Allan et al. 2003) and deer exclusion zones around households (Bloemer et al. 1990). In addition, small scale deer exclusion is likely to mirror the spatially heterogeneous use of habitat by deer, and we may expect high tick intensity and potential tick-borne disease hotspots in areas that deer consistently avoid.

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